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Nonsurgical Management of Postpartum Arterial Mesenteric Thrombosis: An Exceptional Outcome

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Abstract

Acute arterial mesenteric ischemia is exceptional in the postpartum period, and the few published cases mainly describe proximal occlusions of the superior mesenteric artery with a poor prognosis. We report the case of a 37-year-old woman with hypertensive nephropathy and chronic kidney disease who underwent cesarean delivery at 38 weeks of gestation for superimposed preeclampsia. On postpartum day 15, eight days after discontinuation of enoxaparin thromboprophylaxis, she presented with sudden abdominal pain out of proportion to the physical findings, severe high anion gap metabolic acidosis, and acute-on-chronic kidney injury (creatinine clearance 18.7 mL/min). Computed tomography angiography showed partially occlusive filling defects in distal segmental branches of the superior mesenteric artery, with a patent main trunk and preserved bowel wall enhancement. After surgical consultation, conservative management was undertaken in the intensive care unit with intravenous unfractionated heparin, aspirin, correction of metabolic disturbances, and close monitoring against predefined criteria for surgical conversion. Control angiography on day eight of treatment was normal. Treatment was switched to reduced-dose apixaban plus aspirin. The etiological workup revealed isolated anti-SSA/Ro52 antibodies without evidence of lupus and could not formally exclude antiphospholipid syndrome. No recurrence or bleeding occurred during eight months of follow-up. This case suggests that early computed tomography angiography, performed despite renal impairment, can identify distal, pre-necrotic arterial lesions that may be managed conservatively under close surveillance in carefully selected patients.

Categories: Obstetrics/Gynecology
Keywords: acute mesenteric ischemia, anticoagulation, anti-ro52 antibody, postpartum period, superior mesenteric artery

Introduction

Acute mesenteric ischemia (AMI) is a sudden interruption of intestinal blood flow of arterial (embolic or thrombotic), venous, or non-occlusive origin, which leads to bowel necrosis if perfusion is not rapidly restored [1,2]. It is a vascular emergency with a mortality rate that remains close to 50%, mainly because of delayed diagnosis [1-3]. Arterial occlusions usually occur in older patients with an embolic cardiac source or atherosclerosis. Pregnancy and the postpartum period are prothrombotic states, but most cases of mesenteric ischemia reported in this setting are venous [4].

Postpartum arterial mesenteric ischemia has been reported only sporadically [4-7]. The documented cases involved the trunk or proximal portion of the superior mesenteric artery (SMA) and ended in bowel surgery or death. Proximal occlusions interrupt flow to most of the small bowel and right colon, whereas distal branch occlusions may be partly compensated by the collateral network of the mesenteric arcades [3]. We report a distal, segmental thrombosis that was recognized before necrosis and resolved without revascularization or resection in a patient with chronic kidney disease (CKD), a condition that shaped every therapeutic decision. We also discuss the finding of isolated anti-SSA/Ro52 antibodies and its implications for the patient and her child.

Case Presentation

History and pregnancy

A 37-year-old woman (gravida 5, para 2; weight 55 kg) had been followed for five years for hypertensive nephropathy with CKD and was receiving antihypertensive treatment. Her two previous term pregnancies were uncomplicated and ended in scheduled cesarean deliveries, without gestational hypertension or other complications. She had had two spontaneous miscarriages at two months of gestation. She had no personal or family history of thrombosis or connective tissue disease and did not smoke.

The unplanned pregnancy was complicated by superimposed preeclampsia, diagnosed on a systolic blood

pressure of up to 160 mmHg and dipstick-positive proteinuria, both controlled with alpha-methyldopa; prepregnancy blood pressure and proteinuria values were not available, and serum creatinine remained stable at approximately 180 µmol/L throughout pregnancy. Iron deficiency was documented in the third trimester (serum ferritin 9 ng/mL). Fetal growth and amniotic fluid volume were normal. A cesarean delivery was performed at 38 weeks of gestation; a live infant weighing 2,750 g was born with an Apgar score of 10 at one and five minutes. The postoperative course was uneventful, and she was discharged home on postoperative day 3 with oral amoxicillin 2 g/day for seven days. Thromboprophylaxis with enoxaparin 40 mg daily was given for seven days after delivery, with no documented adjustment to renal function.

Acute episode and diagnostic assessment

On postpartum day 15, the patient was readmitted through the emergency department with sudden, stabbing abdominal pain rated 9/10, with vomiting but without cessation of stool or flatus. She was agitated; her heart rate was 105 beats/min, blood pressure 130/60 mmHg on treatment, respiratory rate 24 breaths/min, oxygen saturation 99% on room air, and temperature 37°C. Urine output was preserved. The abdomen was diffusely tender without guarding or rigidity; the cesarean scar was clean, the uterus was well involuted, lochia was minimal, and the cervix was closed. The discrepancy between the intensity of the pain and the paucity of physical signs immediately raised suspicion of mesenteric ischemia.

Admission laboratory tests (Table *1*) showed an inflammatory response (white blood cell count 15,650/mm^3, C-reactive protein 67.5 mg/L) and severe metabolic acidosis (pH 7.20; bicarbonate 8.3 mmol/L) with a high anion gap (at least 20 mmol/L, calculated from bicarbonate and normal sodium and chloride values), normal potassium, and a lactate level of 2.2 mmol/L. Serum lipase was 546 IU/L. Serum creatinine was 321.7 µmol/L, corresponding to a Cockcroft-Gault creatinine clearance of 18.7 mL/min, reflecting acute-on-chronic kidney injury. Coagulation tests drawn before therapeutic anticoagulation showed a prothrombin ratio of 44.8%, an international normalized ratio (INR) of 1.62, and a prolonged activated partial thromboplastin time (aPTT) of 37.9 seconds.

Parameter	Unit	Reference range	Admission	One month	Two months
Hemoglobin	g/dL	12.0–16.0	10.5	9.4	9.7
White blood cells	/mm³	4,000–10,000	15,650	5,300	7,000
Platelets	/mm³	150,000–400,000	343,000	139,000	291,000
C-reactive protein	mg/L	< 5	67.5	1.41, then 3.60	-
Arterial pH	-	7.35–7.45	7.20	-	-
Bicarbonate	mmol/L	22–26	8.3	24	20
Sodium, chloride, potassium	mmol/L	Na 135–145; Cl 98–107; K 3.5–5.0	Normal	-	-
Anion gap	mmol/L	8–16	High	-	-
Lactate	mmol/L	0.5–2.0	2.2	-	-
Lipase	IU/L	13–60	546	-	-
Serum creatinine	µmol/L	45–90	321.7	203.5	199.5
Creatinine clearance (Cockcroft-Gault)	mL/min	> 90	18.7	-	-
Prothrombin ratio	%	70–100	44.8	84.6	-
INR	-	0.8–1.2	1.62	1.09	-
aPTT	seconds	25–35 (control 25)	37.9	24.8	-
AST	IU/L	< 35	21	17	-
ALT	IU/L	< 35	70	20	-
Serum albumin	g/L	35–50	-	-	40.2
Spot urine protein	mg/L	< 150	-	-	425.1

TABLE 1: Course of the main laboratory parameters

Admission coagulation tests were drawn before therapeutic anticoagulation.

ALT: alanine aminotransferase; aPTT: activated partial thromboplastin time; AST: aspartate aminotransferase; INR: international normalized ratio.

Because of severe pain and agitation, abdominal ultrasound was not performed. Abdominopelvic computed tomography (CT) angiography was obtained without delay despite renal impairment, as the risk of a delayed diagnosis outweighed that of contrast-associated kidney injury (thin-section arterial and portal venous phases). It showed hypodense, partially occlusive filling defects in distal segmental branches of the SMA, whose main trunk remained patent (Figure *1*). There was no bowel wall enhancement defect, pneumatosis, portal venous gas, bowel obstruction, or ascites. The aorta and celiac trunk were patent, without mural thrombus or dissection, the pancreas was normal, and the appendix was normal. A 48 × 42 × 26 mm collection adjacent to the cesarean scar, with infiltration of the surrounding fat, was interpreted as postoperative.

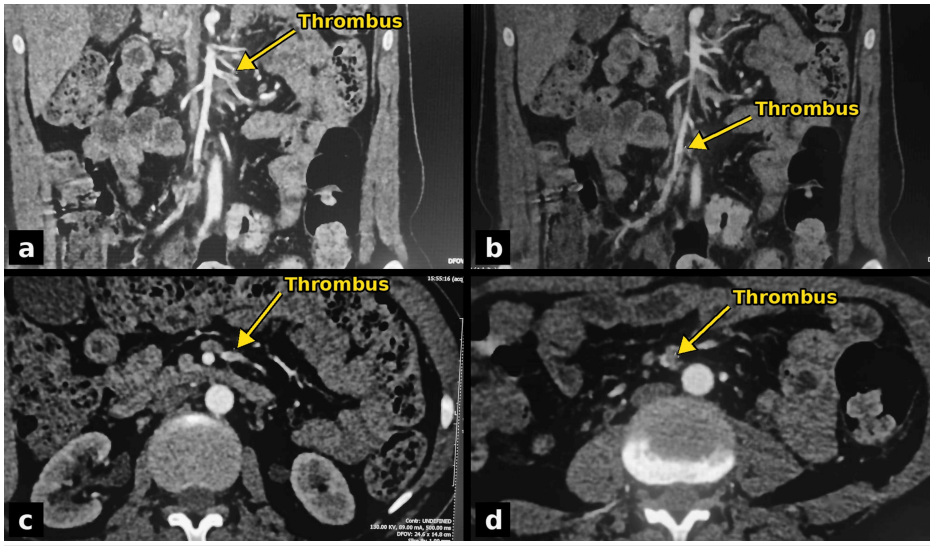


FIGURE 1: Computed tomography angiography of the superior mesenteric artery on admission

Arterial phase. (a, b) Coronal reconstructions; (c, d) axial images. Labels and arrows indicate hypodense, partially occlusive filling defects in segmental branches. The main trunk of the superior mesenteric artery is opacified and patent.

The electrocardiogram showed sinus tachycardia at 110 beats/min, low-voltage QRS complexes, and a corrected QT (QTc) interval of 490 ms, without atrial fibrillation or ischemic changes (Figure 2). Bedside transthoracic echocardiography performed by the consulting cardiologist was reported as normal, with no intracardiac thrombus, valvular disease, atrial dilatation, or regional wall motion abnormality; images were not archived. Holter monitoring was not performed.

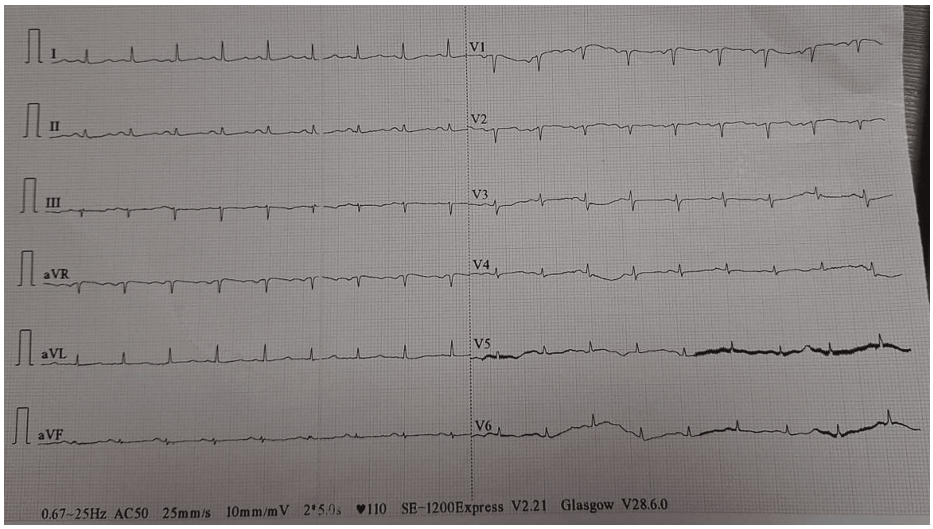


FIGURE 2: Twelve-lead electrocardiogram on admission

Paper speed 25 mm/s, 10 mm/mV. Sinus tachycardia at 110 beats/min, PR 140 ms, QRS 68 ms, normal axis, QT/QTc 362/490 ms (Bazett). Low-voltage QRS complexes and poor R-wave progression from V1 to V4, without ST-segment elevation or Q waves. Artifacts in V5 and V6 are due to patient movement.

The etiological workup is detailed in Table 2. Anti-SSA/Ro52 antibodies were strongly positive (100 U/mL; cutoff 10 U/mL), and all other antinuclear antibody specificities were negative. Complement levels were normal. Anticardiolipin and anti-β2-glycoprotein I antibodies were negative on a single immunodot assay, and lupus anticoagulant was not tested. Antineutrophil cytoplasmic antibodies and anti-glomerular basement membrane antibodies were negative. Parathyroid hormone was elevated at 239.8 pg/mL, consistent with secondary hyperparathyroidism. Inherited thrombophilia testing was not performed.

Test	Result	Interpretation
Anti-SSA/Ro52 (immunodot, 25 antigens)	100 U/mL (positive > 10)	Strongly positive, isolated
Other antinuclear specificities (including anti-dsDNA, anti-Sm, anti-RNP, anti-Ro60, anti-SSB, anti-Scl-70, anti-centromere, myositis-specific antibodies)	0 U/mL	Negative
Complement C3/C4	1.67/0.42 g/L	Normal
Anticardiolipin and anti-β2-glycoprotein I (immunodot)	0 AU (< 5)	Negative; single test, assay not validated for classification
Lupus anticoagulant	Not performed	Not interpretable on heparin
ANCA (MPO, PR3); anti-GBM	0 AU	Negative
Inherited thrombophilia	Not performed	Limitation of the report
Electrocardiogram	Sinus tachycardia; QTc 490 ms	No atrial fibrillation
Transthoracic echocardiography	Normal	No embolic source
Intact parathyroid hormone	239.8 pg/mL (15–68.1)	Secondary hyperparathyroidism
Serum ferritin	61 ng/mL (9 in the third trimester)	Normal

TABLE 2: Etiological workup

ANCA: antineutrophil cytoplasmic antibodies; anti-GBM: anti-glomerular basement membrane antibodies; AU: arbitrary units; dsDNA: double-stranded DNA; MPO: myeloperoxidase; PR3: proteinase 3; QTc: corrected QT interval.

Treatment

The patient was admitted to the intensive care unit (ICU). The general surgeon found no indication for surgery in the absence of clinical or radiological signs of bowel compromise, and the vascular surgeon found no indication for revascularization, given the patent trunk and the distal, partial nature of the lesions. Criteria for conversion were defined on admission: clinical deterioration, peritoneal signs, rising lactate, organ failure, or progression of the lesions or loss of bowel enhancement on control CT angiography.

Intravenous unfractionated heparin (UFH) was started as soon as the CT diagnosis was confirmed (80 IU/kg bolus followed by 18 IU/kg/h). It was preferred to low-molecular-weight heparin (LMWH) because creatinine clearance was below 30 mL/min and because it could be rapidly reversed if surgery became necessary. Monitoring was based on aPTT, with a target of 1.5-2.5 times the control value. Treatment also included aspirin 100 mg daily, multimodal analgesia, bowel rest, fluid resuscitation, and hemodynamic optimization without vasopressors, with repeated abdominal examination, blood gas analysis, and lactate measurement. The acidosis resolved with fluid resuscitation alone, without bicarbonate or renal replacement therapy. No antibiotic therapy was given, as there was no fever, peritoneal sign, or CT evidence of bowel compromise. The antithrombotic strategy is summarized in Table *3*.

Phase	Treatment	Rationale	Monitoring and precautions
After cesarean delivery (postpartum days 0–7)	Enoxaparin 40 mg daily SC	Venous prophylaxis after cesarean delivery	Not adjusted to renal function
Acute phase (from postpartum day 15)	IV UFH: 80 IU/kg bolus, then 18 IU/kg/h	Creatinine clearance < 30 mL/min; reversibility if surgery is needed	aPTT 1.5–2.5 times control; platelet count
Acute phase, then three months	Aspirin 100 mg daily	Arterial component of the thrombosis	Bleeding risk of the combination; limited duration
Discharge, six months	Apixaban 2.5 mg twice daily	Limited renal elimination; no injections or INR monitoring	Off-label; dose-reduction criteria derived from atrial fibrillation; breastfeeding discontinued; reassessment after repeat antiphospholipid testing

TABLE 3: Antithrombotic strategy adapted to chronic kidney disease

aPTT: activated partial thromboplastin time; INR: international normalized ratio; IV: intravenous; SC: subcutaneous; UFH: unfractionated heparin.

Outcome and follow-up

The pain resolved within a few days and bowel function returned; the conversion criteria were never met. Control CT angiography on day eight of treatment showed no residual filling defect, patent SMA branches, and a normal bowel wall. After nephrology and cardiology consultation, the patient was discharged on apixaban 2.5 mg twice daily for six months and aspirin 100 mg daily for three months; breastfeeding had been discontinued beforehand. At one month, she was asymptomatic, serum creatinine was 203.5 µmol/L, and the platelet count was 139,000/mm³, which normalized to 291,000/mm³ at two months; this thrombocytopenia occurred after the switch from heparin to apixaban, and anti-platelet factor 4 antibodies were not tested. After discharge, she was referred to a rheumatologist for the anti-SSA/Ro52 antibodies; no immunomodulatory treatment was started, and repeat antiphospholipid testing is planned during this follow-up. After eight months of follow-up, there has been no thrombotic recurrence or bleeding complication, and the patient is followed in nephrology and rheumatology.

Discussion

Comparison with published cases

Published cases of postpartum arterial mesenteric ischemia involved the trunk or the proximal portion of the SMA (Table *4*). Ducarme et al. reported a proximal thrombosis treated with thrombectomy, ileal resection, and ileostomy [5]. Singhal et al. described a death shortly after cesarean delivery [6], and Gaujal et al. reported a thrombosis of the SMA trunk two weeks after cesarean delivery [7]. Moustaide et al. reported extensive mesenteric infarction associated with a thrombus of the descending aorta, which was found at exploratory laparotomy to be unsalvageable, with death on day four [4]. The only favorable outcome with conservative treatment involved a dissection, not a thrombosis, of the SMA [8]. Some cases initially classified as arterial proved to be venous on histology [9]. A literature search of PubMed and Google Scholar, without language restriction, combining the terms "mesenteric ischemia", "mesenteric artery thrombosis", "postpartum", "puerperium", and "cesarean", identified no case of postpartum arterial mesenteric thrombosis that resolved without revascularization or resection. This difference is explained mainly by the distal level of involvement and the pre-necrotic stage at diagnosis, which made a noninvasive strategy possible.

Reference	Age	Timing, mode of delivery	Involvement	Treatment	Outcome
Singhal et al., 2005 [6]	22	Immediate, cesarean	Not specified	Not specified	Death
Ducarme et al., 2008 [5]	38	Day 8, vaginal	Proximal SMA	Thrombectomy, ileal resection, ileostomy	Survival
Gaujal et al., 2015 [7]	29	Two weeks, cesarean	SMA trunk	Not specified	Not specified
Moustaide et al., 2025 [4]	36	Day 10, vaginal	Proximal SMA, aortic thrombus	Exploratory laparotomy; no resection because of extensive necrosis	Death on day 4
Present case	37	Day 15, cesarean	Distal segmental branches, partial occlusion, patent trunk	UFH then apixaban, aspirin, ICU monitoring	Resolution; no recurrence at eight months

TABLE 4: Postpartum arterial mesenteric ischemia reported in the literature

The SMA dissection reported by Suzuki et al. is not included.

ICU: intensive care unit; SMA: superior mesenteric artery; UFH: unfractionated heparin.

Differential diagnosis

Mesenteric ischemia is not the first diagnosis to consider in acute abdominal pain after cesarean delivery. Surgical-site complications and uterine scar dehiscence were unlikely given the clean scar, the absence of peritoneal signs, and a collection interpreted as postoperative; endometritis was unlikely given the well-involuted uterus, minimal lochia, closed cervix, and absence of fever. CT angiography excluded ovarian vein thrombosis, bowel obstruction or perforation, and appendicitis, and showed a normal pancreas despite hyperlipasemia. The combination of pain out of proportion to the physical findings, metabolic acidosis, and leukocytosis therefore led directly to CT angiography, which showed the arterial filling defects.

Mechanism and predisposing conditions

The postpartum period combines hypercoagulability, stasis, and endothelial injury, which can be aggravated by cesarean delivery and hypertensive disorders of pregnancy [10]. CKD adds endothelial dysfunction, platelet activation, and a procoagulant state, with arterial and venous thrombotic risk increasing as the glomerular filtration rate declines [11]. The severe high anion gap metabolic acidosis with only mildly elevated lactate points first to acidosis of acute-on-chronic kidney injury, with serum creatinine rising from a stable baseline of approximately 180 µmol/L to 321.7 µmol/L, although an ischemic contribution cannot be excluded; serum albumin, glucose, and ketones were not measured at admission. It is a reminder that a normal lactate level does not exclude mesenteric ischemia [1,3]. Hyperlipasemia, common in mesenteric ischemia and increased by renal failure, should neither lead to a mistaken diagnosis of pancreatitis nor delay CT angiography [12]. The admission coagulopathy, with a decreased prothrombin ratio and a prolonged aPTT in the absence of liver failure, remains unexplained, as fibrinogen, D-dimer, and coagulation factors were not measured. Because the baseline aPTT was already prolonged, aPTT-based monitoring of UFH may have overestimated the anticoagulant effect. Finally, although no cardiac or aortic source was found, the absence of Holter monitoring and the multifocal nature of the lesions do not allow in situ thrombosis to be distinguished from distal embolism.

Anti-Ro52 antibodies and antiphospholipid syndrome

Anti-SSA/Ro52 antibodies, directed against the TRIM21 protein, are found in Sjögren's disease, systemic lupus erythematosus, inflammatory myopathies, and systemic sclerosis, but may also occur in isolation. They are not an established thrombotic risk factor and cannot be considered the cause of the event. In the absence of clinical or serological criteria for Sjögren's disease or systemic lupus erythematosus, this finding does not establish a specific diagnosis and is regarded here as an associated finding of uncertain significance. Combined with a nephropathy diagnosed at the age of 32 and two miscarriages, however, they warrant a rheumatological evaluation, which is ongoing. Anti-Ro52 antibodies have also been associated with acquired QT prolongation in adults [13], which calls for reassessment of the QTc of 490 ms, measured here during tachycardia and acidosis. From an obstetric standpoint, these antibodies expose the child to neonatal lupus and congenital heart block [14]: an electrocardiogram of the child is warranted, and any future pregnancy would require fetal echocardiographic surveillance and discussion of hydroxychloroquine.

Antiphospholipid syndrome (APS) has not been excluded. Testing relied on a single immunodot assay, a technique not accepted by the classification criteria, and lupus anticoagulant was not tested, whereas the criteria require standardized tests that remain positive at least 12 weeks apart [15,16]. Repeat testing is planned during rheumatological follow-up, at a time when the patient is not receiving a direct oral anticoagulant, as these drugs interfere with lupus anticoagulant testing. This question has therapeutic consequences: direct oral anticoagulants are not recommended in thrombotic APS, particularly after arterial thrombosis [17], and confirmed positivity would require switching to a vitamin K antagonist.

Rationale for conservative management

Guidelines recommend immediate anticoagulation in all cases of arterial AMI, revascularization, preferably endovascular, for occlusions with ischemia, and surgical exploration in the presence of peritonitis or necrosis [1,2]. They do not specify the management of partial distal lesions without bowel compromise. In our patient, an intervention was not expected to improve flow, as the trunk was patent and bowel perfusion was preserved, whereas it carried specific risks (Table *5*): ischemia-reperfusion injury [18], splanchnic vasoconstriction and a procoagulant state related to surgical stress, uncertain intraoperative assessment of bowel viability that may lead to unnecessary resection or repeated laparotomy [2], and the risks of anesthesia in the setting of severe acidosis, decompensated renal failure, and coagulopathy. Conservative treatment was acceptable only with continuous ICU monitoring, explicit conversion criteria, control CT angiography, and immediate access to surgery. The rapid regression of the lesions suggests that they were treated at a still reversible stage, although a single case does not allow the outcome to be attributed to any one component of management.

Option	Main risk	Relevance in this patient
Exploratory laparotomy	Surgical stress: splanchnic vasoconstriction, hypovolemia, ileus, procoagulant state	No clinical or radiological sign of necrosis
Surgical embolectomy or thrombectomy	Ischemia-reperfusion injury	Patent trunk and preserved perfusion; distal branches difficult to access
Bowel resection	Viability difficult to assess; resection of viable segments; intestinal failure	Bowel perfused on CT angiography
Endovascular thrombectomy or thrombolysis	Additional contrast load; distal embolization; bleeding with fibrinolysis	Creatinine clearance 18.7 mL/min; cesarean delivery 15 days earlier; abdominal wall collection
General anesthesia	Instability related to acidosis; worsening renal function; bleeding; interruption of heparin	pH 7.20; decompensated renal failure; INR 1.62
Conservative treatment	Failure to recognize progression to irreversible ischemia	Continuous monitoring, conversion criteria, control CT angiography

TABLE 5: Possible invasive options and their risks in this patient

CT: computed tomography; INR: international normalized ratio.

Antithrombotic therapy and renal impairment

Renal impairment guided the choice of antithrombotic agents (Table *3*). UFH was suited to the acute phase because of its reversibility and lack of renal accumulation; however, given the prolonged baseline aPTT, anti-Xa monitoring would have been preferable. In retrospect, enoxaparin prophylaxis at 40 mg daily had not been adjusted to renal function, which supports systematic assessment of renal function before any postpartum LMWH prescription. Apixaban was chosen because of its limited renal elimination (approximately 27%) and its favorable safety profile compared with warfarin [19]. These data, however, come from atrial fibrillation; the ARISTOTLE trial excluded patients with creatinine clearance below 25 mL/min, and the dose-reduction criteria applied here (weight ⩽ 60 kg and serum creatinine ⩾ 133 µmol/L) are those of that indication [19]. This was therefore off-label use, to be reassessed according to the results of repeat antiphospholipid testing. Breastfeeding had to be discontinued, as direct oral anticoagulants are not recommended during lactation, unlike heparins and warfarin [20]. Finally, postpartum prophylaxis targets venous events [20], and this case does not show that longer prophylaxis would have prevented an arterial thrombosis.

Limitations

This report has several limitations: it describes a single case; there was no histological confirmation; the

mechanism was not established; inherited thrombophilia testing was not performed, and antiphospholipid testing was incomplete; Holter monitoring, fibrinogen and D-dimer measurements, and serum albumin, glucose, and ketone measurements at admission were not performed, and the exact anion gap could not be calculated; prepregnancy values were unavailable, which weakens the diagnosis of superimposed preeclampsia; proteinuria was measured on a spot sample; and follow-up was limited to eight months. The favorable outcome does not demonstrate the efficacy or safety of conservative treatment in other forms of arterial mesenteric ischemia.

Conclusions

Arterial mesenteric ischemia should be considered in any postpartum patient with severe abdominal pain out of proportion to physical findings, even after thromboprophylaxis and even when lactate is only mildly elevated, and CT angiography should not be delayed solely because of renal impairment, although this decision must be individualized according to the urgency of diagnosis and the available alternatives. In this case, partially occlusive distal segmental lesions without bowel compromise resolved with anticoagulation and close ICU monitoring against predefined criteria for surgical conversion. This strategy can only be considered in carefully selected patients with immediate access to surgery. Renal impairment requires adjustment of every antithrombotic agent, for both prophylaxis and treatment, and the finding of anti-Ro52 antibodies, together with the incomplete exclusion of APS, underlines the importance of a complete and repeated etiological workup.

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.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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