SUNTEXT REVIEWS

SunText Review of Case Reports & Images
ISSN: 2766-4589

Open Access
Case Report
Volume 7:2

Post-Cesarean Uterine Scar Dehiscence Associated with Severe Puerperal Sepsis: A Case Report with Pathophysiological Analysis

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Received date: 10 April 2026; **Accepted date:** 16 April 2026; **Published date:** 22 April 2026

Citation: Villalobos Rodriguez AL (2026) Post-Cesarean Uterine Scar Dehiscence Associated with Severe Puerperal Sepsis: A Case Report with Pathophysiological Analysis. SunText Rev Case Rep Image 7(2): 181.

DOI: https://doi.org/10.51737/2766-4589.2026.181

Abstract

Uterine scar dehiscence following cesarean section is an uncommon but potentially life-threatening complication, often presenting with nonspecific symptoms that may delay diagnosis and treatment. It represents a significant clinical challenge due to its association with severe intra-abdominal infection and maternal morbidity. We report the case of a 34-year-old woman who presented nine days after cesarean section with fever, abdominal pain, and purulent wound discharge. Clinical and imaging findings were suggestive of intra-abdominal infection. Exploratory laparotomy revealed uterine scar dehiscence with myometrial necrosis and intra-abdominal abscess. A total hysterectomy was performed following damage-control principles. The patient had a favorable clinical recovery with intensive care support and antibiotic therapy. Uterine scar dehiscence should be suspected in postpartum patients with persistent infection unresponsive to standard treatment. Early diagnosis and timely surgical intervention are essential to reduce maternal morbidity and mortality.

Keywords: Postpartum sepsis; Uterine dehiscence; Cesarean section; Abdominal abscess; Hysterectomy

Introduction

Post-cesarean uterine scar dehiscence is an uncommon but clinically significant cause of secondary postpartum hemorrhage, reported in less than 1% of women and estimated to occur in approximately 1 in 365 cesarean deliveries [1-4]. The term "uterine dehiscence" generally refers to a progressive disruption of the myometrium without complete transmural rupture; however, it is frequently used interchangeably with "uterine rupture," which may lead to diagnostic ambiguity. This condition is most commonly described in the lower uterine segment following cesarean delivery, where it may also be referred to as incisional dehiscence and may present as either partial or complete separation [5]. Despite its rarity, post-cesarean scar dehiscence may mimic more common postpartum conditions, including infection or retained placental tissue, which can delay diagnosis. Early recognition requires a high index of clinical suspicion due to the potential for rapid clinical deterioration [1]. Reported incidence of early puerperal uterine scar dehiscence ranges from 0.06% to 3.8%, reflecting variability in diagnostic criteria and clinical presentation across studies [6,7]. We present the case of a patient who developed severe puerperal sepsis secondary to uterine scar dehiscence, illustrating the diagnostic challenges and surgical implications of this rare complication. Clinical manifestations suggestive of infection appeared nine days after a low-segment cesarean section performed for dystocia of presentation with ruptured membranes.

Case Report

A 34-year-old woman, gravida 1 para 1, was admitted to the emergency department nine days after undergoing a cesarean section performed for breech presentation during active labor with rupture of membranes. She presented with persistent fever, abdominal pain, and foul-smelling purulent discharge from the suprapubic surgical wound, approximately 12 cm in length. The patient reported no history of chronic diseases or known drug

allergies. On physical examination, she was conscious but lethargic and diaphoretic. She appeared markedly pale and febrile (39 °C), hypotension (90/51 mmHg), tachycardia (118 bpm), and normal breath sounds were preserved. The abdomen was distended and painful on deep palpation, with rebound tenderness, decreased bowel sounds, with clinical signs consistent with peritonitis. The surgical wound showed approximately 2 cm of dehiscence, with continuous purulent discharge and inflamed wound edges. Initial laboratory tests revealed leukocytosis (25,600/µL), neutrophilia (94–95%), C-reactive protein of 433.7 mg/L, and procalcitonin of 0.96 ng/mL. Arterial blood gas analysis showed a pH of 7.43, lactate of 0.5 mmol/L, and a normal anion gap.

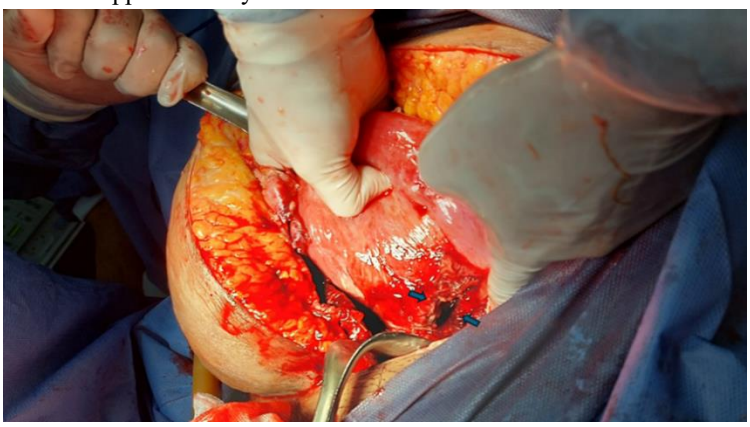

Figure 1: *Uterine defect in the lower segment identified during exploratory laparotomy: finding of dehiscence of previous cesarean section scar with irregular and avascular borders (indicated by the arrow).*

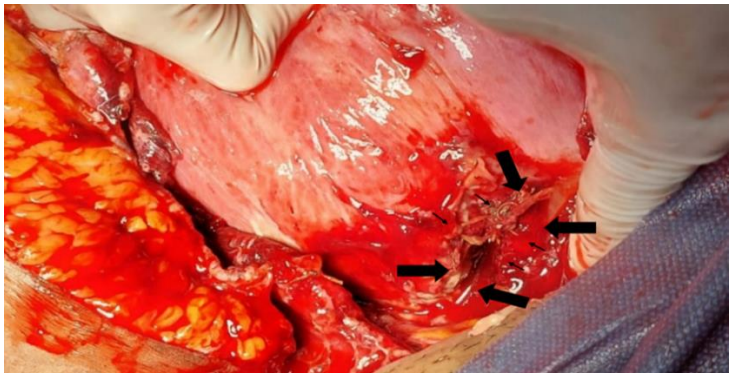

Figure 2: *Magnified view of the dehiscent defect in the lower uterine segment: direct exposure of the necrotic area and devitalized margins at the site of previous cesarean section (indicated by the arrow).*

Figure 3: *Protrusion of edematous intestinal loops at the time of laparotomy, compatible with peritonitis secondary to puerperal sepsis, which conditioned the temporary closure of the abdomen.*

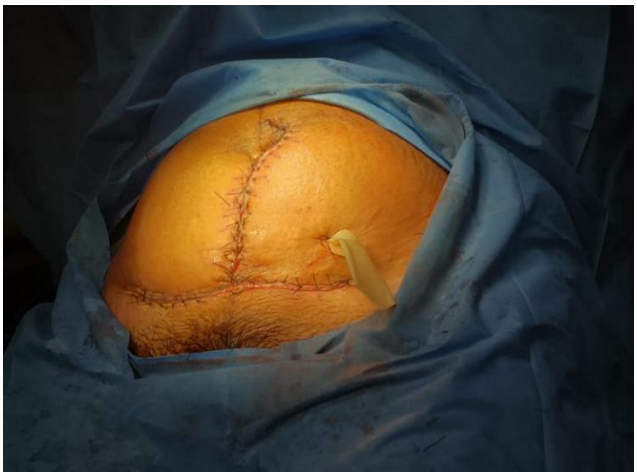

Figure 4: *Immediate postoperative abdomen after subtotal hysterectomy and peritoneal lavage, with flat closure and placement of Penrose drainage for control of residual collection.*

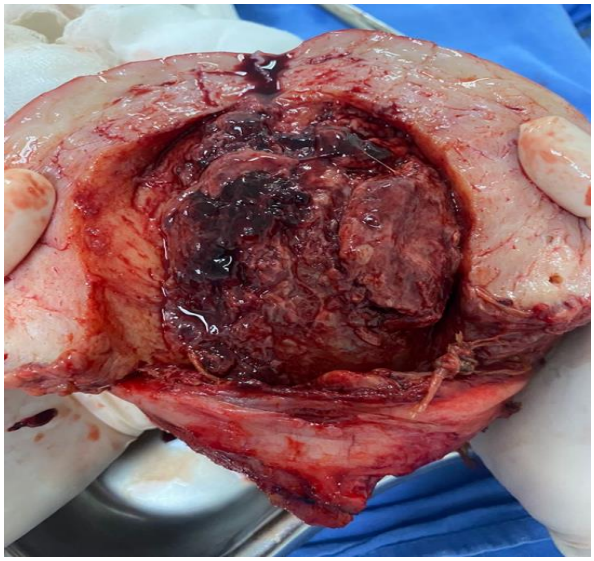

Figure 5: *Macroscopic findings of post-hysterectomy uterus that show loss of continuity solution in the hysterotomy scar, with the presence of necrotic tissue and placental remains adhered to the endometrium, correlated with histopathological diagnosis of acute endometritis with neutrophilic infiltrate, microabscesses and necrotic chorionic villi.*

Table 1: *Temporal evolution of inflammatory and hematologic laboratory parameters during hospitalization.*

Day	Leukocytes (×10³/µL)	Platelets (×10³/µL)	C-reactive protein (mg/L)	Procalcitonin (ng/mL)
Reference range	**4.0–10.0**	**150–450**	**<5**	**<0.05**
1	25.6	473	–	2.1
2	23.7	509	433.7	0.96
3	18.9	496	589.5	0.93
4	18.6	540	252.7	0.84
6	15.4	560	180.5	0.22
10	13.2	941	148.4	<0.05
11	13.7	977	103.5	<0.05
This table illustrates the temporal evolution of leukocyte count, platelet count, C-reactive protein, and procalcitonin during hospitalization. An initial marked inflammatory response, characterized by leukocytosis, thrombocytosis, and elevated biomarkers of infection, was followed by a progressive decline in inflammatory markers, consistent with clinical improvement and therapeutic response. Reference ranges are included to aid interpretation of laboratory trends.				

Table 2: *Comparative analysis of reported management strategies in complicated post-cesarean uterine scar dehiscence: literature review.*

Author / Year	Age / Sex	Main Clinical Presentation	Management Strategy	Indications Described	Procedures Performed	Reported Evolution
Mraihi 2023 [1]	35 / Female	Secondary hemorrhage with uterine dehiscence	Conservative + selective surgical	Initial hemodynamic stability	Antibiotics, uterine evacuation	Favorable resolution without hysterectomy
Bharatam 2015 [2]	25 / Female	Abdominal wound sepsis	Surgical	Extensive wound infection	Debridement + antibiotics	Progressive recovery
Ali 2019 [3]	30 / Female	Late uterine rupture	Radical surgical	Acute abdomen with systemic involvement	Laparotomy and hysterectomy	Favorable evolution
Haridas 2021 [4]	28–37 / Female (case series)	Puerperal sepsis with intra-abdominal abscess	Combined management	Persistent fever and abdominal pain	Laparoscopy/laparotomy with drainage and uterine repair	Clinical resolution
Chawanpaiboon 2024 [5]	28 / Female	Severe puerperal sepsis with uterine dehiscence	Early radical surgical	Peritonitis, necrosis, abscess formation	Total abdominal hysterectomy + drainage	Maternal survival with recovery
Pavoković 2020 [6]	35 / Female	Post-cesarean peritonitis due to hysterorrhaphy dehiscence	Surgical control	Acute puerperal abdomen, endomyometritis	Relaparotomy, necrectomy, uterine resuture	Favorable recovery
Choden 2022 [7]	28 / Female	Abscess and severe sepsis	Radical surgery	Dehiscence with pelvic abscess and infection	Relaparotomy, drainage, uterine resuture	Complete recovery
Badr 2017 [9]	25 / Female	Incisional necrosis	Selective conservative	Stable patient without severe sepsis	Localized resection + antibiotics	Uterine preservation
Shi 2022 [10]	36.5 ± 2.0 / Female	Post-cesarean sepsis (risk factors)	Prevention and surveillance	High BMI, prolonged surgery	Protocol-based management	Reduced complications
Ishikawa 2024 [11]	25.8 ± 0.7 / Female	Early uterine dehiscence	Ultrasound follow-up	Post-cesarean evaluation	3D ultrasound assessment	Early identification
Chavan 2020 [12]	26 / Female	Septic abdomen	Surgical	Persistent fever and abdominal pain	Laparotomy	Clinical resolution
El-Agwana 2018 [13]	20–25/ Female	Localized uterine infection	Conservative	Absence of peritonitis or shock	IV antibiotics	Recovery without major surgery
This table summarizes previously published cases and series describing post-cesarean uterine dehiscence complicated by infection, hemorrhage, or intra-abdominal sepsis. It compares patient characteristics, clinical presentation, therapeutic approach, surgical indications, and outcomes. Overall, the literature shows a spectrum of management ranging from conservative treatment in hemodynamically stable patients to radical surgery, including hysterectomy, in cases associated with peritonitis, necrosis, or severe systemic compromise. The comparison contextualizes the present case within current evidence and highlights decision-making patterns reported in similar clinical scenarios						

Ultrasound demonstrated a deep intra-abdominal abscess with intermuscular edema. Given the suspicion of complicated intra-abdominal infection and signs of impending organ dysfunction, urgent surgical management was indicated. An infra- and supraumbilical exploratory laparotomy was performed. Intraoperative findings included disruption of the uterine scar continuity, necrosis of the hysterotomy edges, avascular margins, refractory uterine atony, and myometrial myonecrosis (Figures 1,2). Approximately 200 mL of purulent fluid was evacuated, extending from the uterine cavity into the subcutaneous tissue and peritoneal cavity. A total obstetric hysterectomy was performed, along with abscess drainage and peritoneal lavage with 2 L of normal saline. Due to marked intermuscular edema (Figure 3), temporary abdominal closure was performed. The patient was transferred to intensive care for hemodynamic support and broad-spectrum antimicrobial therapy. Following the initial surgery, the patient showed partial clinical improvement, with decreased purulent exudate. Two days later, a planned second-look surgery was performed for peritoneal lavage and definitive abdominal wall closure. Resolution of the intermuscular edema was observed, with loose intestinal adhesions and no active bleeding or purulent collections. Secondary closure of the abdominal wall was completed, and broad-spectrum antibiotics were continued (Figure 4).

Subsequent clinical evolution was satisfactory under close monitoring, with progressive reduction of inflammatory markers and adequate wound healing. Culture of purulent material grew Enterococcus faecalis, sensitive to vancomycin and linezolid, and resistant to ciprofloxacin, levofloxacin, and gentamicin. Treatment with imipenem was initiated due to severe postoperative infection with risk of polymicrobial sepsis, requiring broad-spectrum coverage against Gram-positive, Gram-negative, and anaerobic organisms, particularly in the setting of uterine dehiscence and intra-abdominal abscess formation. Histopathological examination revealed a mixed inflammatory infiltrate with acute predominance, characterized by abundant neutrophils infiltrating and destroying the endometrial epithelium, forming microabscesses within glandular lumina. Mature chorionic villi with extensive necrosis, hemorrhage, and inflammation were observed firmly adherent to the endometrium, findings consistent with acute endometritis. Loss of continuity of the hysterotomy scar was also confirmed (Figure 5). During clinical evolution, laboratory parameters reflected an initial systemic inflammatory response followed by progressive recovery. C-reactive protein increased from 433.7 mg/L on admission to a peak of 589.5 mg/L the following day, with a gradual decrease to 103.5 mg/L by February 7. Procalcitonin declined from 2.1 ng/mL to normal values (<0.05 ng/mL). Leukocytosis decreased from 25.6 × 10^3/µL to 13.7 × 10^3/µL, while platelets increased reactively from 473,000 to 977,000 × 10^3/µL. Renal function and electrolyte levels remained within normal ranges (Table 1). Clinical timeline: Low-segment cesarean section for breech presentation during active labor with rupture of membranes (day 0). Admission to the emergency department with fever, abdominal pain, and purulent wound discharge, consistent with septic abdomen (day 9 post-cesarean). Ultrasound confirmed intra-abdominal abscess, prompting urgent laparotomy with identification of uterine scar dehiscence, myometrial necrosis, and performance of total obstetric hysterectomy with temporary abdominal closure (day 9). Planned reoperation with peritoneal lavage and definitive closure was performed 48 hours later, followed by favorable clinical evolution with progressive reduction of inflammatory markers under intensive antimicrobial therapy.

Discussion

Biological and functional basis of post-cesarean uterine healing

In the uterus, healing of the post-caesarean hysterotomy does not occur in a quiescent state, but in the midst of a biologically turbulent postpartum period: the organ is involuting, the myometrium maintains sustained contractions, the hormonal environment changes abruptly (estrogen and progesterone levels drop) and the maternal metabolism tends towards a more catabolic profile; all of which modifies the microenvironment of the wound and, therefore, the quality of the repair. In their expert review, Bujold and Romero emphasize that closing the uterus is not a routine step, but a surgical procedure with long-term biological consequences: the scar must restore continuity, perfusion and tissue alignment, but also preserve reproductive function, where endometrial re-epithelialisation is key to avoiding defects such as niches/isthmoceles [8]. Histologically, the scar tends to be more fibrous (collagen) and with limited smooth muscle regeneration, which explains why the scarred segment rarely regains the strength of the intact myometrium; hence the remodeling phase lasts for months (even up to a year) and short interpregnancy intervals are associated with a higher risk of complications, because the collagen is still reorganizing and the scar has not yet "matured" mechanically.

Risk factors, pathophysiology, and surgical management of complicated post-cesarean uterine dehiscence

Post-cesarean scar dehiscence is a rare but serious cause of sepsis and secondary postpartum hemorrhage, often characterized by an insidious and potentially life-threatening clinical presentation. In exceptional cases, this condition is associated with necrosis and infection of the uterine incision [1,5,7,9,10]. Several predisposing risk factors have been described, including diabetes, emergency

surgery, intraoperative infection, suture technique, hematoma at the uterine incision site, and retrovesical hematoma formation [1,6,7]. Badr reported a series of 23 cases of necrosis and infection of the post-cesarean uterine incision, identifying endomyometritis and postoperative hematoma formation as major risk factors, particularly after emergency cesarean deliveries performed during labor and in the presence of ruptured membranes [8]. Shi conducted a case-control study involving 408 women who underwent cesarean delivery and identified independent risk factors associated with postoperative sepsis. These included elevated body mass index (BMI) (OR = 2.06; 95% CI: 1.23–3.43; p = 0.006), surgical duration longer than 60 minutes (OR = 2.34; 95% CI: 1.39–3.95; p = 0.001), and blood loss exceeding 400 mL (OR = 1.87; 95% CI: 1.12–3.13; p = 0.017) [10]. Similarly, rupture of membranes ≥12 hours (OR = 2.01), labor duration ≥8 hours (OR = 2.67), urinary catheter use ≥24 hours (OR = 2.79), and lack of antibiotic prophylaxis (OR = 2.16) were significantly associated with increased risk of post-cesarean sepsis. Preoperative anemia (Hb <100 g/L; OR = 2.08) and leukocytosis (>10 × 10⁹/L; OR = 2.31) were also identified as contributing factors [10]. In the present case, the main predisposing factors included emergency cesarean delivery performed in the setting of ruptured membranes and active labor, conditions known to increase the risk of postoperative infection and surgical complications. Some authors have also suggested that rapid uterine involution in the immediate postpartum period may negatively affect suture integrity, weaken the scar and predispose to early dehiscence [11].

Among surgical factors associated with postoperative complications, excessively tight sutures during hysterorrhaphy may induce ischemia and focal myometrial necrosis, triggering a localized inflammatory response that, particularly in the presence of anemia, may progress to localized peritonitis. In this context, locking or anchored sutures may increase the risk of tissue ischemia; therefore, non-locking unidirectional sutures have been recommended, especially in patients with predisposing conditions or early signs of tissue compromise [4,6,9]. Ishikawa compared polydioxanone (PDS) sutures with polyglactin 910 barbed sutures in cesarean deliveries, evaluating uterine scar integrity using three-dimensional ultrasound on postpartum day two in 54 women. Dehiscence occurred more frequently in the PDS group (44%) than in the polyglactin group (17.2%) (p = 0.035), with greater defect width (2.2 mm vs. 1.1 mm; p = 0.048), suggesting increased scar vulnerability associated with barbed sutures [11]. Early dehiscence has also been linked to subsequent development of niches (isthmoceles). Uterine dehiscence may create direct communication between the uterine and peritoneal cavities, facilitating translocation of pathogenic microorganisms from the upper genital tract into the peritoneal space and increasing the risk of peritonitis and sepsis. Reported pathogens include Escherichia coli, Klebsiella pneumoniae, Streptococcus spp., and Bacteroides fragilis, among other Gram-negative, Gram-positive, and anaerobic organisms [4]. The clinical presentation may occur two to three weeks after delivery and, in some cases, may be observed up to six weeks later [7,9,11]. In a review of 23 cases, the most frequent clinical manifestations were abdominal pain and persistent fever despite antibiotic therapy for more than 48 hours, with symptom onset occurring between 2 and 15 days after cesarean section in most patients and between 6 and 10 weeks in a smaller proportion [3,9]. In the present case, peritonitis and abdominal distension—findings consistent with previously reported cases of uterine scar dehiscence and infection—developed nine days after delivery. Several studies indicate that ultrasound is the first-line imaging modality for the evaluation of suspected uterine rupture [1,3-5,7,9,12]. In cases of uterine wound sepsis associated with necrosis and endomyometritis, the indication for peripartum hysterectomy has been reported in approximately 6% of patients. Given this clinical and surgical context, hysterectomy was performed as a definitive intervention to control infection and achieve adequate hemostasis [6-9]. Although surgical management is required in patients with severe infection or peritonitis, conservative treatment with broad-spectrum antibiotics and targeted drainage may be effective in hemodynamically stable patients without active bleeding or advanced infection, allowing progressive resolution and favorable clinical outcomes [13].

Physiopathological analysis: rupture of previous uterine scar as a trigger for bacterial sepsis

Uterine rupture over a previous cesarean section scar may represent not only a mechanical event but also a trigger for systemic bacterial dissemination from a previously contained focus. Recent studies have shown that the lower uterine segment (LUS), particularly after cesarean delivery, has a vulnerable anatomical and functional architecture characterized by reduced muscle fiber density, decreased vascularization, and progressive thinning of the myometrium in scarred areas [14-16]. This structure is especially prone to the formation of defects such as niches or isthmoceles, which may act as reservoirs for blood, mucus, or secretions, favoring an anaerobic microenvironment and persistent bacterial colonization [17-19]. Microbiological evidence suggests that the lower uterine segment, even in the absence of overt clinical infection, may harbor significant bacterial colonization after delivery, with predominance of Gram-positive cocci and Gram-negative bacilli [19-21]. These findings are supported by histopathological analyses of uterine niches demonstrating atrophic endocervical epithelium, chronic fibroblastic reaction, and disorganized capillary networks without signs of acute inflammation, supporting the hypothesis of latent uterine dysbiosis [18,19]. The type of suture material used during

cesarean section may also play a role. Absorbable multifilament sutures, such as catgut or polyglycolic acid, have demonstrated greater bacterial adherence and retention compared with monofilament materials because of their capillarity and porosity [22,23]. Consequently, the scar line within the lower uterine segment may become a surface susceptible to biofilm formation on fibrotic tissue or foreign material, remaining clinically silent but vulnerable to activation following structural disruption. In this context, rupture of a sub clinically colonized uterine scar defect may act as a trigger for massive bacterial release. Structural disruption may permit abrupt translocation of microorganisms from the endometrial cavity into the peritoneal space, triggering peritonitis and fulminant sepsis even in the absence of preceding fever or clear clinical signs. Although this sequence has not been validated in controlled clinical studies, converging anatomical, microbiological, and histopathological evidence supports its biological plausibility and highlights the need for targeted research. Future studies analyzing peritoneal fluid, scar niche cultures, and bacterial typing could help validate this hypothesis.

This proposed mechanism may explain clinical scenarios in which no overt signs of infection are initially identified but severe sepsis develops following uterine rupture. It also underscores the importance of considering uterine microbiota, surgical closure quality, and suture characteristics as key factors in the prevention of post-cesarean infectious complications [17-20,22,23]. Comparative analysis of published cases suggests that management of post-cesarean uterine dehiscence exists along a therapeutic continuum primarily determined by the extent of tissue damage and the degree of systemic infection. Patients with localized infection and hemodynamic stability may benefit from conservative strategies based on antibiotics and targeted drainage, whereas myometrial necrosis, peritonitis, or severe sepsis generally require radical surgical management following damage-control principles, including obstetric hysterectomy. Moreover, the literature indicates that many cases evolve from nonspecific initial presentations to septic abdomen, emphasizing the importance of early clinical suspicion and timely imaging. Collectively, these findings support the concept that outcomes are influenced not only by the presence of dehiscence but also by the interaction between bacterial colonization, uterine closure quality, and host inflammatory response—elements consistent with the pathophysiological model proposed in the present case (Table 2).

Conclusion

Dehiscence of the post-cesarean uterine scar leading to puerperal sepsis and peritonitis is a rare but clinically significant obstetric complication, often characterized by an insidious presentation and low initial clinical suspicion. The present case illustrates how a combination of clinical and surgical factors, including emergency cesarean section, prolonged rupture of membranes, suture technique, and possible subclinical bacterial colonization of the lower uterine segment, may result in a severe clinical condition that poses a life-threatening risk. From a pathophysiological perspective, rupture of a previously colonized uterine scar may act as a trigger for massive bacterial dissemination, providing a potential explanatory model for certain forms of fulminant puerperal sepsis. This hypothesis, supported by anatomical, microbiological, and histopathological evidence, suggests that the quality of surgical closure, the type of suture material used, and the structural condition of the lower uterine segment should be considered critical factors not only in the prevention of dehiscence but also in the modulation of infectious risk. Timely surgical intervention, together with intensive multidisciplinary support, allowed a favorable clinical outcome, underscoring the importance of early recognition and coordinated management. Prospective studies are needed to further evaluate the relationship between scar defects, bacterial colonization, and post-cesarean sepsis, as well as the role of imaging modalities and biomarkers in early detection. Integration of these elements into obstetric surgical protocols may contribute significantly to reducing maternal morbidity in similar clinical settings.

Ethical Considerations

This work was reviewed and evaluated by the Research Committee CI-2025-063-HGC and Research Ethics Committee CEI-2025-063-HGC of the General Hospital of Cancun Dr. Jesus Kumate Rodriguez

Conflicts of Interest

There are no conflicts of interest.

Sources of Funding

There are no sources of funding.

This case was conducted in accordance with CARE guidelines. Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

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