CASE REPORT

Early uterine rupture mimicking pancreatitis in the early second trimester of pregnancy: A case report

Saira Bhatti[1], Sana Ashfaq[2], Shakira Parveen[3]

Abstract

The case of a 28-year-old woman with prior caesarean deliveries, who came in with severe abdominal pain along with 18 weeks of gestation, is presented. Ultrasound showed a live intrauterine pregnancy accompanied with oedematous pancreas and mild to moderate fluid in the abdomen. Despite supportive care, her condition deteriorated with dropping haemoglobin and worsening abdominal pain. Computed tomography (CT) scan suggested possible appendicitis or focal uterine rupture. Despite conservative management her pain persisted. Repeat ultrasound revealed a uterine wall defect with placenta protruding through it, suggestive of uterine rupture. Emergency laparotomy confirmed uterine rupture at the previous uterine scar, with placenta percreta invading the bladder wall. During the procedure, the patient had an estimated blood loss of 2,000ml. After receiving antibiotics and blood transfusions, she recovered and was discharged after 21 days. This case highlights a rare but life-threatening complication of a second-trimester uterine rupture in a scarred uterus.

Keywords: Placenta accreta syndrome, Spontaneous uterine rupture, Second-trimester, Placenta percreta.

DOI: https://doi.org/10.47391/JPMA.21380

Introduction

Uterine rupture is an obstetric emergency associated with significant maternal-foetal mortality and morbidity. It can lead to substantial maternal bleeding and severe uterine injury which may require a hysterectomy.[1] Uterine rupture refers to a disruption of all uterine layers including the serosa while uterine dehiscence is an incomplete uterine scar separation with intact serosa.[2] Second trimester spontaneous uterine rupture occurring between 13 and 28 weeks of gestation is a very rare event with an estimated prevalence of 1 in 5,000 to 20,000 pregnancies.[3] The ratio of scarred uterus rupture to unscarred uterus rupture is approximately 3:1.[4] The main risk factors for a uterine rupture include grand multiparity, obstructed or neglected labour, injudicious use of oxytocin and previous caesarean section, short inter-pregnancy interval, and abnormal placentation.[4,5]

Department of General Surgery, Recep Tayyip Erdogan Hospital, Muzaffargarh, Pakistan.

Correspondence: Maryam Sana Ehsan Ullah. e-mail: sanaehsan436@gmail.com
ORCID ID: 0009-0008-0975-1640

Submission completed: 13-07-2024 **1st Revision received:** 25-10-2024
Acceptance: 08-11-2025 **Last Revision received:** 07-11-2025

Case Report

A 28-year-old woman (gravida 4, para 2+1), who was 18 weeks pregnant, was brought to the surgical assessment unit of Dr Ruth K. M. Pfau Civil hospital, Karachi, on March 3, 2024, with one-day history of sudden onset severe upper abdominal pain. She had no associated symptoms. On physical examination, her abdomen was soft, tender, and non-distended, with generalised tenderness that was severe in the epigastrium and right hypochondriac region.

The patient's past medical and surgical history was significant for two previous caesarean deliveries and one miscarriage three years back.

On arrival her vital signs were: blood pressure 110/72 mmHg, pulse 103 bpm, respiratory rate 18 breaths/min, and temperature 36.6°C. Initial laboratory tests showed haemoglobin of 9.6 g/dl (normal 12–15.5 g/dl), haematocrit of 27.4% (normal 36–47%), total leukocyte count of 31.2 x10^9/L (normal 4–11 x10^9/L), neutrophils of 87%. Abdominal and pelvic ultrasound revealed a single, viable intrauterine pregnancy corresponding to 18 weeks of gestation with an altered liver texture, swollen and oedematous pancreas, and mild to moderate fluid in the abdomen.

The patient was initially referred to the medical emergency department with a suspected diagnosis of acute pancreatitis on March 4, 2024. However, her condition continued to deteriorate, with a drop in haemoglobin from 9.6 to 6.4g/dl requiring transfusion of two units of packed red blood cells. Her C-reactive protein increased from 18.7 to 66.1 mg/L (normal <10 mg/L). The abdominal pain became worse and localised to the right inferior quadrant with positive rovsing, psoas, and obturator signs on abdominal examination.

Due to the worsening clinical status a whole abdomen computed tomography (CT) scan was performed. The CT scan findings (Figure 1) were suggestive of a swollen, fluid-

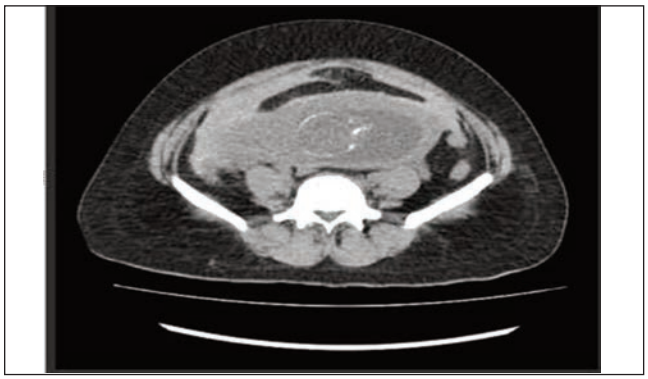

Figure: Whole abdomen computed tomography scan with contrast show hyper dense collection adherent to the anterior abdominal wall and the uterus measuring approximately 1.8x18x9.8 cm marked by red arrows)

filled appendix with minimal surrounding fat stranding as well as an ill-defined, hyper dense collection in the right iliac fossa, which was adherent to the anterior abdominal wall and the uterus measuring approximately 1.8x18x9.8cm (anteroposterior x transverse x craniocaudal) with a volume of 158ml. Gravid uterus with indistinct anterior wall was noted. The pancreatic tail appeared slightly bulky with minimal surrounding fat stranding and minimal thickening of the left lateroconal fascia. Serum amylase and lipase levels were advised to rule out sub-acute pancreatitis which were within normal limits. Overall, the imaging appearances were suggestive of ongoing infective/inflammatory process with possibility of ruptured appendix; however, the possibility of focal uterine rupture could not be entirely excluded. Surgical and obstetric review was advised.

The patient was admitted to the surgery ward for close monitoring and observation. She was initiated on intravenous (IV) antibiotics and ringer's lactate solution. Her vital signs remained stable throughout her hospital stay, and she remained afebrile. Despite blood transfusions, her haemoglobin persisted below normal, and her abdominal pain continued to worsen. Since the pain did not resolve, a pelvic ultrasound was advised on March 9, 2024. The repeated ultrasound demonstrated an intrauterine alive pregnancy corresponding to 18 weeks ±1 week of gestation. A defect was observed in the anterior uterine wall at the site of the scar, measuring approximately 3.1cm, with the placenta seen protruding through it, suggestive of uterine perforation with an organised collection adjacent to it, measuring 13.2x2.5x10.2 cm, with approximately 187 ml.

The patient underwent hysterectomy, right internal iliac artery ligation, and abdominal packing on March 9, 2024. Intraoperative findings revealed haemoperitoneum of approximately 1,500 ml. There was a scar line rupture extending through the entire thickness of the uterine wall. The placenta was partially adherent to the uterine cavity and protruding out, and it was completely adherent and invading the lower uterine segment and bladder serosa (placenta percreta). The estimated blood loss during the procedure was approximately 2,000 ml. In the post-operative period, the patient experienced intermittent fever spikes which were managed with a course of intravenous antibiotics. After an appropriate treatment course and an uneventful recovery, the patient was discharged from the hospital on March 22, 2024 without any follow-up appointments.

Discussion

Placenta accrete spectrum refers to a range of abnormal placental implantation characterised by the abnormal invasion of placental villi into the myometrium. It includes placenta accreta, placenta increta, and placenta percreta.[6] Placenta percreta is the most severe form of abnormal placental implantation where the placental villi penetrates through the myometrium and can invade adjacent structures such as the rectum and bladder.[7]

Women with prior uterine surgery such as myomectomy, curettage, and, in particular, a uterine scar secondary to caesarean delivery have increased risk of placenta percreta.[8] Likewise, this patient had two prior caesarean deliveries which was the likely cause of the placenta percreta.

Abnormal placental implantation such as placenta percreta and a scarred uterus are significant risk factors for uterine rupture.[9] While uterine rupture is most commonly encountered in the third trimester of pregnancy, it can also occur, though rarely, in the second trimester.[4] However, in cases with risk factors, like a scarred uterus in this patient, the possibility of uterine rupture should be ruled out regardless of the gestational age. A comprehensive and detailed history should be obtained from the patient which would help in early recognition and management of this life-threatening obstetric complication.

Patients with uterine rupture can present with a variety of non-specific symptoms, which may mimic other conditions. As highlighted in this case, the patient initially presented with symptoms suggestive of acute pancreatitis or appendicitis without any vaginal bleeding. Regardless of the presenting symptoms, obstetric complications should be considered as one of the top differential diagnosis in pregnant patients.

A live intrauterine pregnancy does not rule out the possibility of a uterine rupture.[10] If the uterine wall and placenta cannot be visualised on ultrasound in a pregnant female presenting with atypical abdominal complaints and

anaemia, further investigations, like magnetic resonance imaging (MRI), can be considered.[8] This is because the inability to fully evaluate the uterine wall on ultrasound, raises concerns about the possibility of a uterine rupture or other complications, even in the presence of a live intrauterine pregnancy.

Conclusion

This case highlights that uterine rupture, although less common, can occur even in the second trimester in women with a prior caesarean scar, and its presentation may mimic non-obstetric conditions such as pancreatitis or appendicitis. Early suspicion and timely imaging are crucial to avoid delay in diagnosis, particularly when laboratory and radiological features are non-specific. This case reinforces the importance of maintaining clinical vigilance when a pregnant woman with a known uterine scar presents with unexplained or worsening abdominal pain.

Disclaimer: The patient provided written informed consent for the publication of this case report. The patient's personal information has been anonymised to protect her privacy.

Conflict of interest: None.

Funding disclosure: None.

References

1. Finnsdottir SK, Maghsoudlou P, Pepin K, Gu X, Carusi DA, Einarsson JI, et al. Uterine rupture and factors associated with adverse outcomes. Arch Gynecol Obstet. 2023;308:1271-78. doi:10.1007/s00404-022-06820-w.
2. Uleanya O, McCallin K, Khanem N, Sabir S. Recurrent uterine rupture in third trimester of pregnancy. BMJ Case Rep. 2021;14:e241987. doi:10.1136/bcr-2021-241987.
3. Ofir K, Sheiner E, Levy A, Katz M, Mazor M. Uterine rupture: differences between a scarred and an unscarred uterus. Am J Obstet Gynecol. 2004;191:425-29. doi:10.1016/j.ajog.2004.01.026.
4. Maymon R, Mor M, Betser M, Kugler N, Vaknin Z, Pekar-Zlotin M, et al. Second-trimester and early third-trimester spontaneous uterine rupture: a 32-year single-centre survey. Birth. 2021;48:61-65. doi:10.1111/birt.12510.
5. Abrar S, Abrar T, Sayyed E, Naqvi SA. Ruptured uterus: frequency, risk factors and feto-maternal outcome: current scenario in a low-resource setup. PLoS One. 2022;17:e0266062. doi:10.1371/journal.pone.0266062.
6. Arakaza A, Zou L, Zhu J. Placenta accreta spectrum diagnosis challenges and controversies in current obstetrics: a review. Int J Womens Health. 2023;15:635-54. doi:10.2147/IJWH.S392002.
7. Jauniaux E, Collins S, Burton GJ. Placenta accreta spectrum: pathophysiology and evidence-based anatomy for prenatal ultrasound imaging. Am J Obstet Gynecol. 2018;218:75-87. doi:10.1016/j.ajog.2017.05.067.
8. Fitzpatrick KE, Sellers S, Spark P, Kurinczuk JJ, Brocklehurst P, Knight M. Incidence and risk factors for placenta accreta/increta/percreta in the UK: a national case-control study. PLoS One. 2012;7:e52893. doi:10.1371/journal.pone.0052893.
9. Savukyne E, Bykovaite-Stankeviciene R, Machtejeviene E, Nadisauskiene R, Maciuleviciene R. Symptomatic uterine rupture: a fifteen-year review. Med. 2020;56:1-7.
10. Abdulwahab DF, Ismail H, Nusee Z. Second-trimester uterine rupture: lessons learnt. Malays J Med Sci. 2014;21:61-5.