Spontaneous uterine rupture at 28 weeks: A case report
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Abstract
Spontaneous Uterine rupture is associated with massive intra-peritoneal bleed which can be fatal if not recognized. We report a case of 32 year old multigravida at 28 weeks of gestation with history of liver cysts, previous caesarean and uterine curettage, who presented with acute abdominal pain and tenderness; ultrasound revealed placenta percreta. CT abdomen showed haemoperitoneum. The patient underwent emergency caesarean hysterectomy due to uterine rupture at the cornual site.

Keywords: Placenta percreta, Ultrasound, Uterine rupture, Computed Tomography.

Introduction
Spontaneous rupture of the non-labouring uterus is a relatively rare occurrence in obstetrics.1 Although cases reported in the literature have described this event in unscarred and primigravid uterus;2 there have been strong associations with abnormal placentation,3 previous uterine instrumentation4 and congenital abnormality of the uterus.5

We report the case of a spontaneous cornual uterine rupture at 28 weeks. Although the patient had a low lying morbidity adherent placenta and previous caesarean section, the site of the rupture, the gestational age and the presentation in this case was unusual and rare.

Case Report
A 32 years old Para 1+1 was booked at 17 weeks of gestation. Patient was a diagnosed case of polycystic kidney disease and Liver cyst. She had a past history of emergency caesarean section in 2011 at 35 weeks of gestation due to anhydramnios and pregnancy induced hypertension. Apart from that, there was also a history of dilatation and evacuation in 2010 due to missed abortion at 10 weeks of gestation.

She presented to the labour room in May, 2014 at 28 weeks of gestation at 1900 hours with complaint of lower abdominal pain on and off since one day. At the time of admission she was stable vitally and pain was generalized. On examination there were no palpable uterine contractions and cervical os was closed. CTG was reactive. She was admitted for the work up of urinary tract infection and rule out threatened preterm labour. IV antibiotics and prophylactic tocolytics were started.

Patient was referred to radiology for ultrasound foetal well-being which showed single viable foetus of 28 weeks gestation. Placenta was left lateral completely covering cervical os and there were multiple bizarre appearing venous lacunae. Doppler examination revealed blood vessels seen at internal os traversing into bladder wall, raising a possibility of placenta percreta (Figure-1).

The patient was subjectively better and there was no evidence of UTI on urinalysis. She was planned for discharge after 48 hours.

On the second day of admission patient had an episode of severe acute abdominal pain at 1130 hours. Her blood pressures dropped to 80/52mm/hg and she had a heart rate of 104/min. There were no uterine contractions and foetal heart sounds were positive. She was resuscitated with fluid while a cause was investigated for the event. The pain was mainly epigastric with rebound tenderness. General surgery input was taken and an abdominal

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Ultrasound and CT scan were advised as there was a suspicion of a ruptured liver cyst. Her amylase, lipase, renal function tests were normal. Liver function tests showed an SGPT of 223 IU/L with a normal bilirubin and other enzymes. Patient's haemoglobin dropped from 11 gm/dl (hematocrit 36) at the time of admission to 9 gm/dl (hematocrit 29).

Ultrasound abdomen showed polycystic liver, bilateral polycystic kidneys with moderate abdominal free fluid having moving internal echoes suggesting haemoperitoneum. CT scan abdomen showed mild high density ascites suggesting intraperitoneal haemorrhage and no definite area of active contrast extravasation was identified. (Figure-2 & 3) Placenta accreta was difficult to visualize on CT due to poor sensitivity and resolution of CT for the myometrium.

The provisional diagnosis was a ruptured liver cyst and the patient was shifted under the services of General Surgery. She was transfused with 2 units of packed red cells.

At 0100 hours the patient again had an acute episode of pain and became haemodynamically unstable. The Obstetrician decided for an emergency caesarean section and the patient was moved to the operating room.

Intraoperatively, there was a haemoperitoneum of 3 liters.

There was uterine rupture at the left cornu with absent anterior uterine wall musculature. A baby boy weight 1400 grams was delivered with an Apgar of 6 at 5 minutes. Due to an absent anterior uterine wall musculature, caesarean hysterectomy was performed. The estimated blood loss was about 3.5 liters and 5 units of packed red cells were transfused.

She remained well in the postoperative phase and recovered smoothly.

The histopathology of the placental specimen showed chorionic villi extending to the superficial myometrium confirming the diagnosis of placenta accreta.

**Discussion**

A previous history of caesarean section is the most frequent cause of uterine rupture. Rupture due to placenta accreta/percreta has also been reported in patients with history of caesarean section and unscarred uterus but mainly in the first and second trimesters. The etiology is a deficient uterine muscle wall in cases with placenta accreta. One might argue that the presence of multiple risk factors (previous scar, previous uterine curettage and placenta accreta) in our patient made this uterus prone to uterine rupture, nevertheless the site of rupture was not previous scar and the patient was not in labour. A history of liver cysts and polycystic kidney was misleading and the decision of caesarean section was

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**Figure-2:** Coronal contrast enhance CT scan showing bilateral polycystic kidneys (a), multiple hepatic cysts (b), and hemoperitoneum (c).

**Figure-3:** Coronal contrast enhance CT scan showing intrauterine foetus with left lateral placenta (a), haemoperitoneum (b), multiple hepatic cyst (c).
initially delayed due to this. Moreover, the diagnosis of rupture was not obvious on radiological examination.

The most valid explanation of this would be the history of previous uterine curettage for missed miscarriage. The reported uterine perforation rate is 19.8 per 1000 at the time of dilatation and curettage. There may have been and unidentified perforation which lead to a scar and hence a weak area in the uterine musculature.

Although the ultrasound and CT scan abdomen showed haemoperitonemium the cornual uterine defect remained undiagnosed and the cause of bleed was mistakenly attributed to the liver cyst. There has been a reported case of diagnosis of a uterine defect in early pregnancy diagnosed on ultrasound which was repaired and the pregnancy continued till 32 weeks. This could be an approach in patients with uterine curettage to diagnose a uterine wall defect.

**Conclusion**

Cases of uterine rupture during labour have been reported in association with previous scar, placenta accreta in first and second trimesters and cornual dehiscence with history of salpingectomy. This case is a rare presentation in third trimester in a non-labouring patient. The main lesson learnt from this case is that the possibility of a potential uterine rupture should always be kept in mind when a gravid woman presents with acute abdominal pain and signs of shock.

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**References**