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# Surgical Management of Placenta Accreta in a Patient with Impending Eclampsia

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## *ABSTRACT*

*A 38 year old lady, with 3 previous caesarean sections, presented to our centre with severe pre-eclampsia. She subsequently underwent an emergency caesarean section at 26 weeks gestation for impending eclampsia and was noted intraoperatively to have placenta accreta. As the morbidly adherent placenta was incidentally detected intraoperatively and was not interrupted during the caesarean delivery, we decided to manage the placenta accreta conservatively by leaving the placenta in-situ. However, in the initial postpartum period, her biochemical markers underwent a sharp deterioration, and a specialist high risk team opinion was sought, with potential plans for return to theatre for a caesarean hysterectomy. The patient subsequently improved after the first 48 hours of close observation, and was discharged well with controlled blood pressures and normal biochemistry on postoperative day 8. She was last reviewed at 10 months postnatal, and is currently doing well with almost complete resorption of the placenta.*

## BACKGROUND

Placenta accreta is an abnormal adherence of placental chorionic villi to the myometrium, which is associated with partial or complete absence of decidua basalis.[1] It has an incidence of about 1 in 1000 deliveries, but has increased 10-fold over the last 50 years due rising caesarean section rates.[1]

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Risk factors of placenta accreta include placenta previa, previous caesarean sections, previous uterine curettage and maternal age greater than 35 years, amongst others.[1,3,4] It has been reported that placenta accreta occurs in 5% of cases of placenta previa with an unscarred uterus, rising to 25% with one previous caesarean section.[2]

Management is controversial. Caesarean hysterectomy, previously considered to be the gold standard of care, is associated with a higher risk of maternal morbidity and mortality. Conservative management, which involves leaving the placenta in situ, has been increasingly sought to reduce maternal morbidity associated with caesarean hysterectomy whilst maintaining fertility. This however, requires protracted monitoring and potentially an interval hysterectomy should complications such as bleeding or infection arise.

While many studies have demonstrated successful conservative management of placenta accreta[1-6], none have described such a management in a patient with concomitant pre-eclampsia, where issues of a retained placenta causing a persistent pre-eclamptic state may be encountered. We describe one such case where conservative management and judicious observation proved to be successful.

## CASE PRESENTATION

A 38 year old lady, para 4, with 3 previous caesarean sections first presented to our centre at 23+1 weeks gestation, having been referred from her private obstetrician's clinic for raised blood pressure of 165 mmHg systolic and 123 mmHg diastolic. Prior to this episode, she had been antenatally well.

With regards to her past obstetric history, she had had 3 previous caesarean sections. The first was an elective caesarean section performed at term for maternal request; the second was an elective repeat caesarean section, also performed at term. These first 2 pregnancies were with her first partner. She had an emergency caesarean section in her third pregnancy for twins at 30 weeks gestation due to severe pre-eclampsia, with her current partner. She denied having any subsequent essential hypertension.

At presentation, her blood pressure was 169 mmHg systolic and 115 mmHg diastolic, with a mean arterial pressure of 121 mmHg. She was asymptomatic and on examination, neurologically intact, with normal reflexes and no clonus. Her symphyseal-fundal height was appropriate for dates and her abdomen was soft and non-tender .Urine dipstick for proteinuria was negative and she was admitted for further investigations and management of her new onset hypertension.

She was started on oral anti-hypertensives (labetalol), and a quantitative 24 hour urine total protein was collected and was detected at less than 0.07g. She was diagnosed with pregnancy induced hypertension without proteinuria. An ultrasound of the fetus was performed during this admission, and the fetus was growing appropriately with adequate liquor volume. The placenta was noted to be low lying and anterior.

She was subsequently discharged after 3 days when her blood pressure was adequately controlled on oral labetalol 200mg TDS. She was advised to measure her blood pressure at home and symptoms to be aware of, and given an early follow up appointment.

However, she returned to our centre at 26+1 weeks with persistently elevated blood pressure readings as well as oliguria. She denied any symptoms of headache, visual disturbances, epigastric pain or right hypochrondrium pain. Fetal movements were felt. Her blood pressure at presentation was 177 mmHg systolic and 112 mmHg diastolic and her mean arterial pressure was at 135 mmHg.

On examination, she was hyper-reflexic, but had no clonus. Her abdomen was soft and non-tender, her uterus was relaxed, and her lungs were clear. Fetal heart rate was detected. A bedside urine dipstick showed proteinuria of 3+. Her anti-hypertensive agents were titrated and she was counseled on the diagnosis of superimposed pre-eclampsia, and the possible need for delivery should her condition deteriorate. She was administered steroids for fetal lung maturity and placed on fluid restriction, with strict observation of her blood pressure and urinary output. Blood investigations performed included a full blood count, a renal panel, liver function test, and coagulation function. Her hemoglobin was 12.3 g/DL, platelet count was 179 $x10^9$/L, creatinine was 60 umol/L, aspartate transaminase (AST) was elevated at 35 U/L, and coagulation panel (PT/aPTT) was normal - PT 11.6s and aPTT 32s. A further 24-hour urine sample was collected for total protein measurement, and was detected to be 5.5g.

Consequently, her blood pressure remained poorly controlled and she required intravenous labetalol. In view of her poorly controlled blood pressure and potential preterm delivery via caesarean section, we also started her on intravenous magnesium sulphate for fetal neuroprotection. A fetal scan was performed to assess for fetal growth restriction. Fetal growth was appropriate for gestation, with an abdominal circumference of 209 mm, and liquor volume was adequate. However, once again, the placenta was noted to be lower and anterior (2 cm from the internal cervical os).

She was reviewed by our specialist high risk Maternal–Fetal Medicine Team the following day and a multidisciplinary team meeting was held with regards to her antenatal care and plans for timing and mode of delivery. The Neonatal Team was also alerted with regards to her admission and potential preterm delivery for severe pre-eclampsia.

The following day, the patient complained of an acute onset of epigastric pain and visual blurring. Her blood pressure was recorded at 140 mmHg systolic and 103 mmHg diastolic, and there was palpable tenderness over her epigastrium. Her reflexes were brisk. Repeat blood investigations revealed an acute elevation of her AST to 406 U/L as well as a drop in her platelet count to 140 $x10^9$/L. Otherwise her hemoglobin was normal at 12.8 g/Dl, creatinine was stable at 56 umol/L, and coagulation profile was normal.

She was deemed to be in severe pre-eclampsia with impending eclampsia and HELLP syndrome. The decision was made for emergency caesarean delivery. In view of her history of 3 previous caesarean deliveries with a known low-lying placenta, the possibility of placenta accreta was discussed with the patient and her partner prior to the caesarean section. Management options of conservatively leaving the placenta in-situ versus a caesarean hysterectomy were discussed. The patient was keen for conservative management, but was aware of the risk of needing a caesarean hysterectomy should there be heavy intraoperative hemorrhage. Blood products were ordered to be delivered to the operating theatre, and the Neonatal Team was alerted to be at hand. There was unfortunately insufficient time to alert our interventional radiology team for pre-operative femoral artery catheterization for uterine artery ligation.

Intraoperatively, the placenta was found to be infiltrating the myometrium of the lower segment of the uterus, but was localized to the uterus, with no bladder involvement. The fetus was delivered through a transfundal incision via breech extraction. A premature fetus weighing 826g was delivered and immediately placed in the care of our Neonatal Team. There was no interruption to the adherent placental mass, and minimal bleeding was encountered. Figure 1 is a picture taken intraoperatively of the adherent placental mass after delivery of the fetus. In view of this, our team made the decision for conservative management by leaving the placenta in-situ, ligating the cord, and closing the uterine incision. The surgery concluded uneventfully, lasting 56 minutes, and the patient was transferred to our high-dependency unit for observation.

However, postoperatively, the patient's biochemical markers took a turn for the worse. Within 5 hours of delivery, her AST level rose from 406 U/L to 2810 U/L, ALT rose from 338 U/L to 1408 U/L and platelet count dropped from 140 $x10^9$/L to 37 $x10^9$/L. There were concerns with regards to the retained placental mass impeding the resolution of her pre-eclamptic state and we sought advice from our specialist high risk Maternal–Fetal Medicine Team. As the patient was clinically asymptomatic, her epigastric pain having resolved after delivery and her blood pressures were under control, the decision was made for careful observation with immediate recourse to uterine artery embolization and a hysterectomy should her blood pressure become uncontrolled or her biochemical markers further deteriorate.

The patient was monitored very closely in our high-dependency unit over the next few days. She remained clinically stable with well-controlled blood pressures. Her biochemical markers were next repeated 10 hours after delivery, and had begun to taper down – her AST decreased from 2810 U/L to 2137 U/L; her ALT decreased from 1408 U/L to 1219 U/L, and her platelet count remained stable from 37 $x10^9$/L to 36 $x10^9$/L. Further repeat tests showed gradual but steady improvement and returned to baseline by postoperative day 6. Table 1. and Chart 1. reflect the changes in levels of biochemical markers as described.

She was discharged well on postoperative day 7, and advised on symptoms to present with, be it elevated blood pressures, symptoms of impending eclampsia, or symptoms of postpartum hemorrhage due to placental separation. She was given a follow up appointment in a specialist outpatient clinic in 2 weeks.

At her outpatient review, she remained clinically stable, and serial serum B-hCG as well as ultrasound measurements of her residual placental mass were performed.

Subsequent outpatient reviews saw her serum B-hCG resolving 15 weeks after her caesarean section. At her last review, at 10 months after delivery, the placental mass had shrunk considerably to only 10.5 x 5mm in measured size. Table 2 and Chart 2 reflect the gradual decrement of the measured size of placental mass over months and Table 3 and Chart 3 reflect the resolution of serum B-hCG over days.

## DISCUSSION

In this interesting case of a pregnancy complicated by both pre-eclampsia and placenta accreta, additional concerns our team had with regards to conservative management of placenta accreta were that of delayed regression of placental function and a persistence of a pre-eclamptic state.

Traditionally, management of placenta accreta mandates a caesarean hysterectomy, which encompasses caesarean delivery of the fetus, followed immediately by a hysterectomy. This measure not only renders a woman sterile, but is also associated with significant maternal morbidity such as massive bleeding, bladder injury and in certain cases, even mortality.

In recent years, there has been a surge in popularity of conservatively managing placenta accreta by leaving the placenta in-situ, which allows for uterine conservation in patients for whom future fertility is desired. This may also precede an interval hysterectomy, which has the advantage of avoiding complications associated with a caesarean hysterectomy[2,5,6,7].
Many centers, including ours, have begun to routinely offer both options of management – caesarean hysterectomy versus leaving the placenta in-situ, to patients with an antenatally detected morbidly adherent placenta[8].

Although placenta accreta was not detected antenatally in our patient due to her late presentation to our centre, a high index of suspicion in view of her having placenta previa, a history of 3 previous lower segment caesarean sections and maternal age greater than 35 years prompted us to carry out a pre-operative discussion with her with regards to the management should placenta accreta be detected intraoperatively. She opted for conservative management i.e. leaving the placenta in-situ, with early recourse to caesarean hysterectomy should severe intraoperative hemorrhage be encountered.

This pre-operative counseling proved to be judicious, as there was indeed intraoperatively detected placenta accreta. Great care was taken not to interrupt the adherent placental mass and there was minimal bleeding encountered intraoperatively. As such, our team made the decision to leave the placenta in-situ.

We expected the patient's pre-eclampsia to resolve with delivery, however, when her biochemical markers deteriorated sharply in the acute postoperative period, we questioned if the retained placenta was impeding her recovery.

The pathogenesis and pathophysiology of pre-eclampsia are still incompletely understood. It has been postulated that defective spiral artery remodeling leads to placental ischemia and results in imbalances between anti-angiogenic and pro-angiogenic factors which cause widespread endothelial dysfunction and finally, affects all maternal organ systems[9].

To date, there is also little literature on how soon the placenta ceases to function after delivery in cases of conservatively managed placenta accreta. There have been a few case series[3,4] suggesting that the hormonal function of the retained placenta declines much more rapidly than visualized tissue regeneration as observed by declining serial serum B-hCG readings. One such article even calculated a half-life of serum B-hCG to be $5.2 \pm 0.26$ days[10].

A search revealed that there has been no reported antecedent clinical scenario where a patient with concomitant issues of preeclampsia and placenta accrete was managed by caesarean delivery and leaving the placenta in-situ.

This case highlights the successful conservative approach to managing placenta accreta in a patient with known preeclampsia. While the patient's biochemical markers did rise in the acute postoperative period, watchful waiting proved to be the appropriate decision she remained clinically well, and her biochemical markers subsequently declined till baseline by a week postoperatively. This reflects that despite being left in-situ, the placental function ceases rapidly after delivery of the fetus and cessation of maternal-placental-fetal circulation. After discharge from hospital, she was well and follow up measurements of her retained placenta and serum B-hCG decreased as expected.

This case further reminds us that the management of complex pregnancies, especially with few antecedent reported cases, requires regular assessment of the patient's clinical condition and individualization of plans. When in doubt, a multi-disciplinary team approach and advice from a specialist high-risk team cannot be further emphasized.

## CONSENT

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

**Figure 1 : Intraoperative picture taken after delivery of baby via transfundal uterine incision. There was no surgical interruption of the anterior accreta (arrow).**

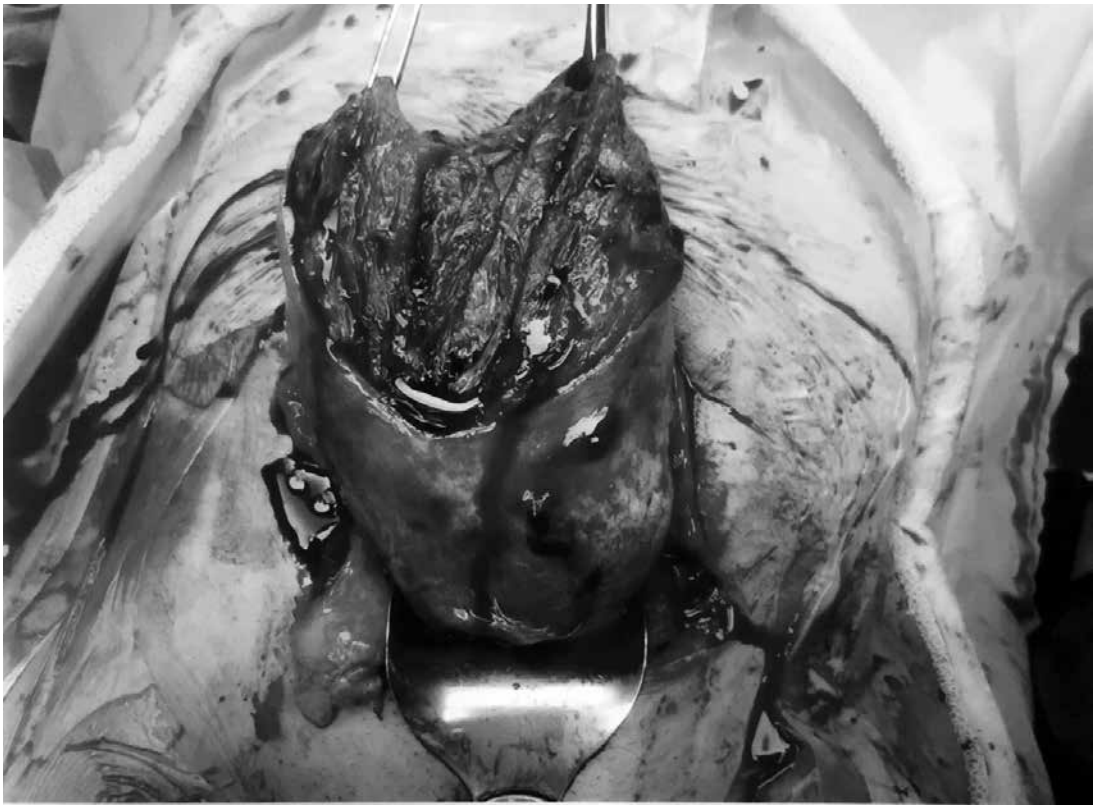

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***Table 1. Table reflecting trend of PE bloods with relation to delivery***

| Relationship to delivery | Right before delivery | 5 hours after delivery | 10 hours after delivery | 14 hours after delivery | POD 1 | POD 1 | POD 2 | POD 3 | POD 4 | POD 6 | POD 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **AST (U/L)** | 406 | 2810 | 2137 | 1374 | 507 | 280 | 207 | 91 | 64 | 29 | 17 |
| **ALT (U/L)** | 338 | 1408 | 1219 | 1045 | 643 | 469 | 409 | 260 | 188 | 95 | 19 |
| **GGT (U/L)** | 160 | 259 | 250 | 222 | 173 | 194 | 202 | 217 | 252 | 204 | 118 |
| **Cr (umol/L)** | 59 | 65 | 69 | 69 | 62 | - | 50 | 51 | 53 | 50 | 56 |
| **Uric Acid (umol/L)** | 484 | 582 | 574 | 555 | 505 | - | 406 | 403 | 399 | 409 | 413 |
| **Plt (10(9)/L)** | 140 | 37 | 36 | 35 | 39 | 52 | 69 | 113 | 142 | 218 | 405 |
| **Hb (g/DL)** | 12.8 | 12 | 11.9 | 10.9 | 9.4 | 9.1 | 10.1 | 9.6 | 9.9 | 901 | 10.5 |
| **PT (sec)** | 11.5 | 14.7 | 15.2 | 15.2 | 13.8 | - | 11.8 | 12.2 | 12.4 | 11.3 | 11.3 |
| **aPTT (sec)** | 27.6 | 34.2 | 34 | 34 | 39.2 | - | 35.9 | 36 | 35.6 | 35.5 | 32.8 |

***Table 2. Table reflecting ultrasound monitoring and resolution of remnant placental mass post-delivery.***

| **Number of days postpartum** | 7 | 40 | 113 | 169 | 299 |
|---|---|---|---|---|---|
| **Size of placental mass on US (mm)** | 58.7 x 57.3 x 43.7 | 64 x 57 x 37 | 36.2 x 34.6 x 23.7 | 12 x 8.9 x 2 | 10.5 x 5 |

***Table 3. Table reflecting monitoring and resolution of serum B-hCG values after delivery.***

| **Number of days postpartum** | 2 | 15 | 40 | 70 | 113 |
|---|---|---|---|---|---|
| **Level of serum B-hCG** | 23943.5 | 10840.1 | 746.2 | 37.4 | <1.2 |