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ROLE OF METHOTREXATE AND UTERINE ARTERY EMBOLIZATION IN A CASE OF PLACENTA ACCRETA

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ABSTRACT

Placenta accreta occurs due to abnormally invasive implantation of placenta. Diagnosis can be made during pregnancy by ultrasound. It may cause severe postpartum hemorrahge that may even require obstetric hysterectomy. We report a case of sonographically diagnosed placenta accreta in a patient with previous 2 cesarean sections. conservative management was attempted with methotrexate and uterine artery embolization. It was not successful and patient started developing features of sepsis. She underwent abdominal hysterectomy. But her blood loss was optimal and she remained haemodynamically stable throughout her hospital stay.

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INTRODUCTION

Placenta accreta has been reported in up to 0.9% of pregnancies and remains a cause of maternal mortality (Yang, 2006). It occurs due to defect in the decidua basalis that results in abnormally invasive implantation of placenta. The anchoring placental villi come in direct contact with the myometrium instead of decidual cells. This defective implantation causes incomplete separation of the placenta during delivery and causes postpartum hemorrhage. The spectrum includes invasion of the superficial myometrium (accreta), invasion into deeper myometrial layers (increta), and invasion through the serosa and/or adjacent pelvic organs (percreta) (Tong, 2008). Traditionally, caesarean hysterectomy at the time of delivery has been the preferred management strategy for placenta accrete (Oyelese, 2006). In some settings, uterine conservation (with the placenta left in situ) may be an alternative strategy (Tong, 2008; Timmermans, 2007; Kayem, 2004; Bretelle, 2007 and Sentilhes, 2010). Adjuvant therapy with methotrexate has also been used to expedite resorption of placental tissue (Timmermans, 2007; Kayem, 2004 and Bretelle, 2007).

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Case Report

A 34 years old G4P3L1 female with 32 weeks of pregnancy with previous 2 cesarean section was admitted on July 24,2017 in SDM hospital, Obstetrics and Gynaecology department with complaints of decreased fetal movements since 1day. It was not associated with pain abdomen, leaking or bleeding per vaginum. Patient's first delivery was a preterm vaginal delivery at 7 ½ months i/v/o preeclampsia in 2007 (died just after birth), second delivery was full term cesarean section i/v/o preeclampsia in 2008 (male child-alive and healthy), third delivery was preterm cesarean section at 6 ½ months i/v/o antepartum hemorrhage in 2015(died just after birth). She was a known case of hypertension since 2007 and was on antihypertensives on and off, hypothyroidism since 1998 was taking tablet eltroxin 100mcg once daily. On admission, patient's pulse rate was 78/min and blood pressure was 130/80 mm Hg. Patient was afebrile with mild degree of pallor. On per abdominal examination, scar of previous sections was noted. Uterus was around 32weeks size, relaxed, cephalic with no localization of fetal heart sound and without scar tenderness. Ultrasonography(USG) showed single 30weeks 6 days IUD fetus in cephalic presentation with placenta in anterior lower segment interrupting myometrium with vascularity in interphase between bladder and myometrium

suggesting possibility of placenta previa/accreta. Investigations revealed haemoglobin of 10.9 gm% with "O"positive blood group. LFT, KFT and urine reports were normal. Coagulation profile was not deranged. Patient was counselled about the complications of adherent placenta and hysterectomy. Patient was undertaken for bilateral uterine artery embolization on same day of admission in order to reduce placental vascularity. She was also given 2doses of 50mg methotrexate intramuscularly on 25/7/17 and 27/7/17 with alternate dose of 5mg folinic acid intravenously on 26/7/17 and 28/7/17 and was started on a course of prophylactic antibiotic. Both these interventions could be done in the patient as she was a diagnosed case of having an IUD fetus. Patient was taken up for hysterotomy by classical section with bilateral tubal ligation on 28/7/17. 1.51kg IUD female was delivered. It was complete placenta previa with placenta accreta. Spontaneous expulsion awaited for 20minutes followed by manual removal. Almost 70-80% of placental mass was removed. She developed vaginal bleeding intraoperatively, so intrauterine packing was done. Intraabdominal drain was placed. 1 unit PRC was transfused intraoperatively with 4 units FFP transfusion postoperatively. Vitals of the patient were stable in the postoperative period.

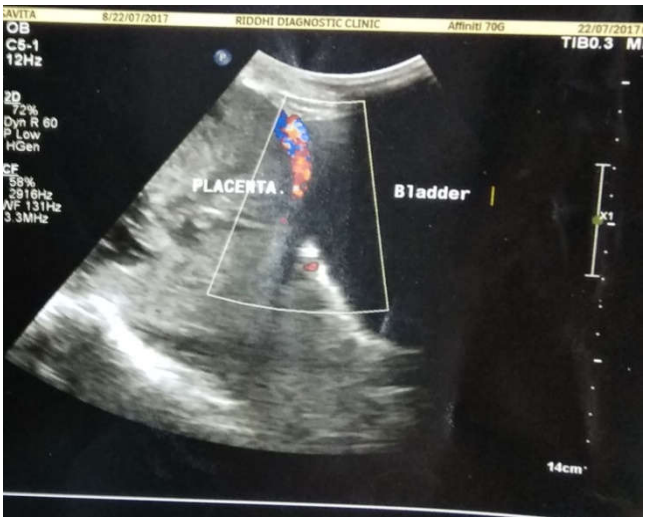

Fig. 1. Ultrasound image showing vascularity at bladder and myometrial interphase

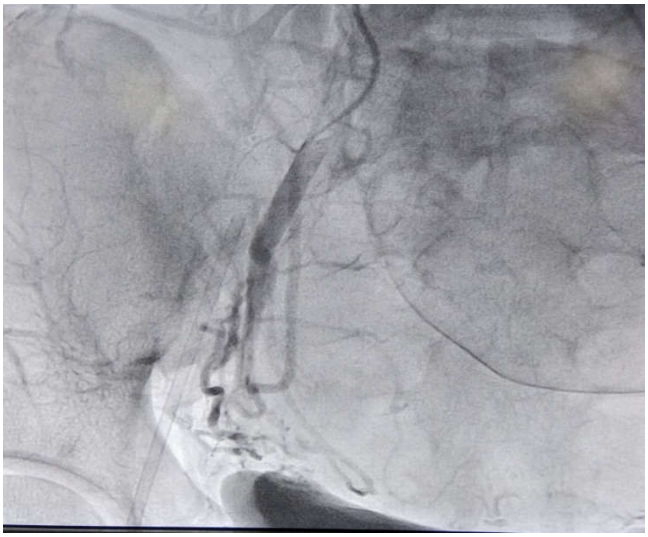

Fig. 2. Placental bed blood flow during start of right side uterine artery embolization

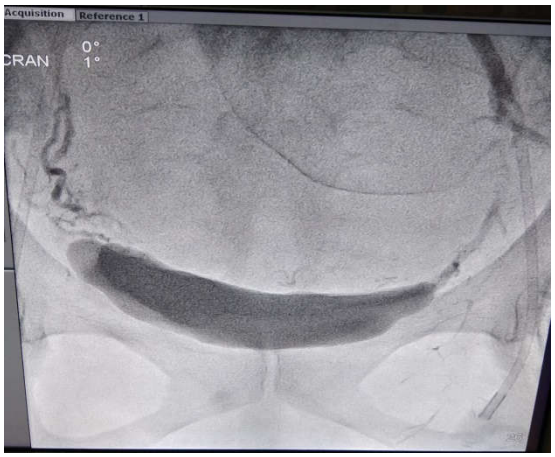

Fig. 3. Placental blood flow after completion of uterine artery embolization

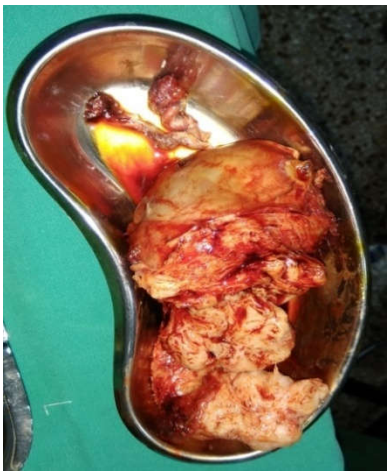

Fig. 4. Specimen of uterus with cervix and bilateral tubes following abdominal hysterectomy

Intauterine packing was removed on postoperative day2 with drain removal on day 4. On the day of discharge her haemoglobin was 8.3 gm% with normal LFT and KFT reports. She was discharged on day 5. Then on postoperative day 11 patient developed on and off fever rising upto 101°F with chills and came for follow up in OPD on day14. on per vaginum examination, os was open with products of conception felt in vaginal cavity. She was admitted again on 10/8/17. Ultrasonography (USG) showed bulky 149x126x85mms sized uterus with dilated endometrial canal. Large adherent placental tissue like structure was seen in whole of uterine body with linera vascular flow at left uteroplacental site suggestive of ?Large RPOC ?? Adherent placenta. Her haemoglobin was 8gm% on 10/8/17. Fever was controlled and vitals stabilized with broad spectrum antibiotics. She was posted for D and E on 11/8/17 with informed consent of hysterectomy if needed. D&E was performed but sudden fresh bleeding was seen coming out thorugh os, so decision for abdominal hysterectomy was taken up. Lower uterine segment was densely adherent to urinary bladder. Hysterectomy was performed successfully in conjunction with urologist. Abdominal drain was placed. She was shifted to ICU for one day. 3units PRC and 4units FFP were transfused postoperatively. Her vitals were stable in postoperative period. Drain was removed on postoperative day3. Her haemoglobin on the day of discharge was 8.7 gm %. She was discharged on 14/8/17 with catheter in situ for 14days.

In follow-up on 14th day, patient was stable and catheter was removed. Pathological report confirmed placenta accreta.

DISCUSSION

The rising incidence of placenta accreta is due to worldwide increase in the number of cesarean sections. It is a life-threatening condition associated with high maternal morbidity and mortality rate being as high as 7% (Resnik, 1999). The risk factors for placenta accreta are previous uterine surgery (like cesarean sections, myomectomy), previous D&E, placenta previa, advanced maternal age, multiparity, Asherman's syndrome and presence of fibroids (Fergal, 1999). Specific and optimal management strategy of abnormally invasive placenta remains unclear. Traditionally, primary hysterectomy at the time of caesarean section has been the mainstay of therapy particularly in cases where the diagnosis has been discovered antenatally (Committee on Obstetric Practice, 2002). In addition to obvious loss of fertility, complications include injury to the gastrointestinal or urinary tracts, infection, as well as massive obstetrical hemorrhage and its sequelae (Committee on Obstetric Practice, 2002). Furthermore, it has been recognized that planned caesarean hysterectomy is associated with fewer perioperative complications compared to emergent procedures (Briery, 2007). Various methods for conservative management of placenta accreta are also emerging these days. Bilateral uterine artery embolization, argon beam coagulation of the placental bed and ligation of uterine artery or anterior division of internal iliac artery has been mentioned with varying success (Das, 2014). It also includes spontaneous placental resorption with injection methotrexate or wedge resection of the area where the placenta is adherent (Das, 2014). Methotrexate acts by causing placental tissue necrosis resulting in rapid involution of residual placenta. This contradicts the belief that methotrexate acts only on rapidly dividing cells, given that trophoblast proliferation is not felt to occur at term (Winick, 1967).

There is no data available on the fixed dosing, frequency or route of administration of methotrexate in such patients. In our studies we used 2 doses of 50mg per m^2 of body surface area with folinic acid 5mg dosing on alternate days similar to the medical management protocol used for ectopic pregnancy. Although conservative management of placenta accreta appears to be successful at preventing hysterectomy in most cases, there is still potential for morbidity. If such an approach is used, intensive monitoring for complications is required. Women may continue to be at risk for weeks to months after delivery (Das, 2014). The various complications include fever, continued vaginal bleeding, secondary haemorrhage, disseminated intravascular coagulopathy and sepsis. Uterine artery embolization has also been tried in our case as another method for conservative management. It led to reduced blood flow to the placental bed thus leading to reduced blood loss during hysterotomy. There is lack of sufficient evidences and studies to highlight the effectiveness of this method and its potentiality needs to be explored.

Conclusion

Conservative management with methotrexate and uterine artery embolization appears as a safe and effective alternative to surgical management in cases of abnormally invasive placenta**.** We report a case of placenta accreta in which both methotrexate and uterine artery embolization were used in order to obliterate the need of obstetric hysterectomy. Despite of their use, the patient got subjected to surgical intervention. But the blood loss was optimal and the patient remained haemodynamically stable. Thus, conservative management of placenta accreta could be justified in selective patients.

Conflict of Interests

Written consent has been taken from the patient described in this paper for its publication. The author has no conflict of interests to disclose.

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