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Early Pregnancy Invasive Placenta – Late Presentation: Case Report and Review of Literature

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Introduction: Placenta percreta is the severe form of morbidly adherent placenta (MAP) where chorionic villi penetrate through the myometrium and to or through the serosa involving bladder or bowel in some cases and can be associated with severe complications.

Case Presentation: In our present case we had a 30-year-old, Gravida 4, Para 2 Living 2 Abortion 1 with previous 2 caesarean sections, present with bleeding on and off since 3 months, post spontaneous abortion at 13+ weeks followed by incomplete check curettage. On examination she was found to have a boggy mass per vagina and a serum Beta-Human Chorionic Gonadotropin (BHCG) value was slightly above normal. On imaging and evaluation she was found to have a mass occupying the lower uterine cavity invading the myometrium with significant vascularity, reported as invasive mole provisionally. She was taken up for laparotomy and hysterectomy after counselling in detail and correction of anemia. Intra-operatively a ballooned out lower uterine segment with a vascular mass was noted which was partially invading the bladder wall. Patient had an uneventful post op period. Her final histopathology report showed placenta percreta with few viable chorionic tissue

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Discussion: Morbidly adherent placenta in early pregnancy can be tricky to diagnose and manage. Patient antenatal history of previous 2 caesarean sections and low-lying placenta in index pregnancy and a failed curettage gave us important clues to her diagnoses along with the MRI findings and BHCG values. Hysterectomy seemed best option for her.
Conclusion: High index of suspicion and planning is required to manage placenta percreta cases successfully.

Keywords: Early pregnancy; invasive placenta; placenta percreta.

1. INTRODUCTION

Morbidly adherent placenta also referred to as *accrete syndromes* is used to describe a spectrum of abnormal placentation and firm adherence due to partial or total absence of decidua basalis and imperfect development of fibrinoid / Nitabuch layer or defect of biological functions of trophoblast leading to excessive invasion. Variants of MAP are classified based by depth of trophoblastic growth. Placenta accreta indicates that villi are attached to myometrium, in placenta increta villi actually invade the myometrium and in placenta percreta villi penetrate through or to the serosa [1] and sometimes to bowel or bladder.

The overall incidence of MAP is rising due to rising number of caesarean section (CS). Placenta percreta accounts to 5-7% of abnormal adherent placenta [2]. The 2 most significant risk factors for placenta accrete are previous CS and placenta previa. An asymptomatic antenatal condition can result in life threatening complications during interventions to separate placenta. Both Magnetic resonance imaging (MRI) and ultrasonography (USG)are modalities for prenatal diagnosis of MAP although limitations exist for both. In our case patient presented with history of spontaneous abortion and incomplete curettage with bleeding on and off for 3 months which made her diagnosis tricky considering the possibility of gestational trophoblastic disorders presenting in similar manner.

2. CASE PRESENTATION

A women with the age of 30 years, Gravida 4 para 2 living 2 abortion 1 with 13+ weeks gestation was referred to us with suspicion of gestational trophoblastic neoplasia for further management. She gave us a history of spontaneous abortion at home 3 months back, followed by attempted check curettage at local hospital along with sterilisation done in the same sitting. However according to records, minimal tissue was obtained on curettage and misoprostol was inserted for retained products and discharged. She however continued to bleed on and off after that and on evaluation was found to have a large boggy mass per vagina when examined by local doctor 2 months later. Her ultrasound pelvis revealed a large lobulated lesion of 9.3 x 6.9 cmx 2.6 cm-260cc with multiple cystic areas and high internal vascularity in the lower uterine cavity with uterus measuring 12.9 x 4.23x 6.85cm. She was transfused 2 pints Packed red blood cell(PRBC) in view of low haemoglobin of 9 and referred here for further management. Further plain MRI done revealed an ill- defined lesion 8.6 x 8.1x 9.2 cm in lower endometrial canal with hemorrhagic areas within. The possible diagnosis was reported as retained products of conception. Serum BHCG was done weekly which revealed serially decreasing values of 61,22.81 and 17 respectively.

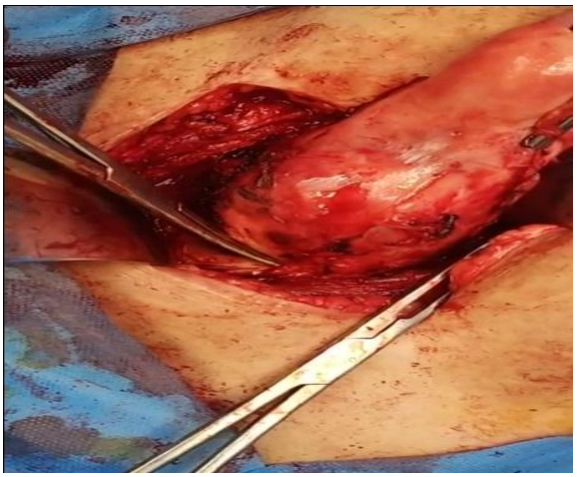
On further evaluation she gave us an obstetric history of previous 2 CSs and a spontaneous abortion at 4 months prior to the present pregnancy. In the present pregnancy she gave a history of on and off bleeding since the time of conception with her Nuchal Translucency-Nasal Bone scan showing a single live intrauterine 13+5 weeks gestation and low lying placenta. No other abnormality was otherwise mentioned. On speculum examination the cervix was found to be to be normal and on per vaginal examination a large boggy mass of 14 weeks size occupying all fornices was felt which was thought to be consistent with uterus and parametrium was found to be free.

On admission, BHCG was repeated in our lab which was found to be 16. MRI with contrast revealed a large well defined uterine lesion of 8.5 x 7.8 x7.6 cm occupying mid and lower uterine cavity which was causing thinning of uterine wall and possibly adherent/infiltrating the wall with possible suspicion of trophoblastic tumour was reported. She was posted for laparotomy and

hysterectomy with a suspicion of gestational trophoblastic tumor /adherent placenta.

Intra op we found the fundus of uterus normal and the lower segment ballooned out with tumour which was very vascular on appearance with bladder completely adherent to the lower segment .We infiltrated the uterus with dilute vasopressin to achieve hemostasis and hysterectomy was carried out. During dissection of bladder adhesions, we noted a small rent of 2 cm in the bladder (region of invasion to bladder) which was then repaired in 2 layers. The patient was transfused one pint PRBC intra op with estimated blood loss of approximately 700 ml. She recovered well in post op period and was discharged in 3 days in stable condition. Catheter was removed on day 14.

Her HPE report: On gross examination: bulky uterus with cervix measuring 12 x 8.5 x 6 cm. On cut section : a polypoidal growth measuring 8 x 7 cm in the lower endometrial cavity with extensive areas of hemorrhage and necrosis involving myometrium and extending upto serosa.Grossly tumor involves serosa. HPE of the polypoidal growth revealed degenerated ghost like chorionic villi infiltrating into the myometrium.Few viable chorionic villi noted.No decidual reaction noted in adjacent areas.Extensive areas of hemorrhage and necrosis present upto serosa. Areas of intermediate trophoblastic proliferation around the blood vessels was noted focally. No hyperplastic trophoblastic cell proliferation seen. cervix was unremarkable. Impression:Features are of invasive/adherent placenta(placenta percreta)



Fig,. 1. **Intra op finding showing ballooned out lower segment**

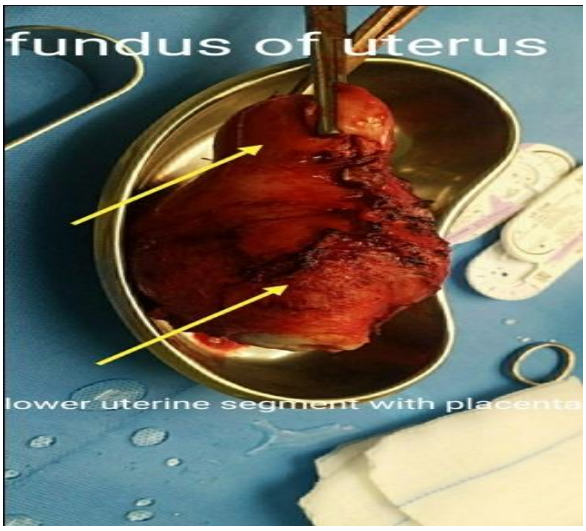


Fig. 2. **Gross specimen**

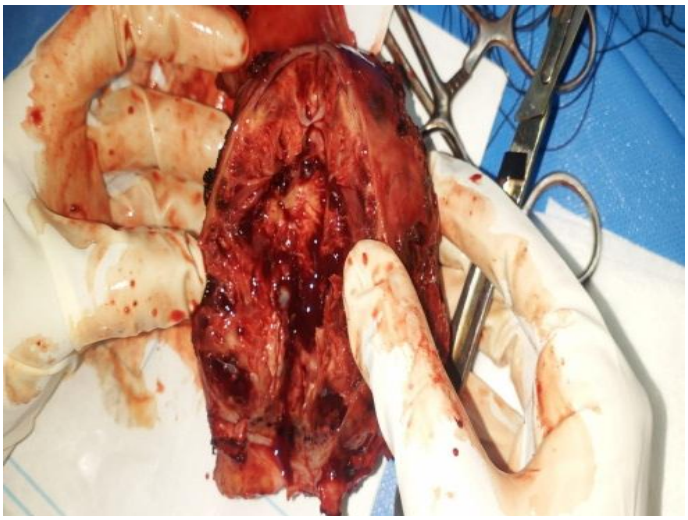


Fig. 3. Cut section of lower uterine segment showing invasion

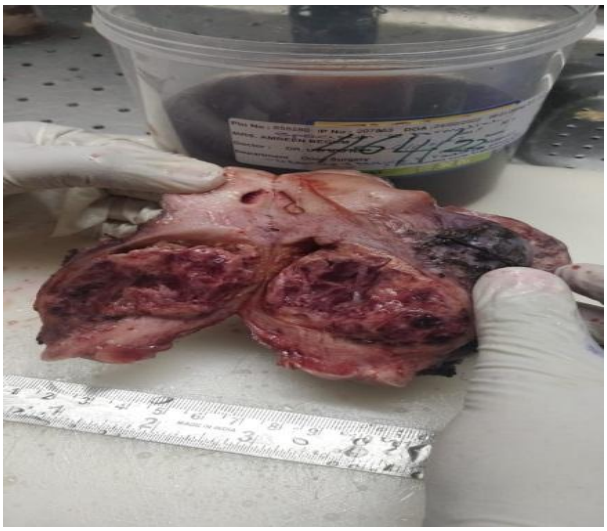


Fig. 4. Pathology specimen on cut section

3. DISCUSSION

In cases of first and second trimester accrete syndromes, there is usually haemorrhage that is a consequence of coexisting placenta previa. Such bleeding will prompt evaluation and management, else it goes on undiagnosed till third trimester. In our case although low lying placenta was picked up on scan, MAP was not recognised. Both USG and MRI are modalities for prenatal diagnosis of MAP although limitations exist for both techniques.3-dimensional power Doppler may complement in the antenatal diagnosis of MAP. Thin decidual endometrium and myometrium, as well as placental extension into the myometrium, are identified. Loss of hypoechoicity of the myometrium between the bladder and the placental wall can be detected using sonograms. Additionally, the presence of intraplacental sonolucent areas, commonly known as venous lakes, next to the implicated uterine wall strongly suggests placenta accrete. With competent sonographers, diagnostic sensitivity and specificity can reach 85 to 90%. If the results of a sonogram are unclear, an MRI can be performed. Accreta can be predicted by signs including uterine bulging into the bladder, heterogeneous signal strength within the placenta, and the existence of intra placental bands. When placenta percreta is suspected, cystoscopy can be done to detect bladder involvement. In our case due to late presentation possibility of invasive mole was also considered.

"Most common predisposing factor of abnormal placentation is previous CS and placenta previa, as was in our case. Other factors are prior uterine scars such as uterine curettage, myomectomy, Asherman's syndrome, iatrogenic uterine perforation, manual removal of the placenta, advanced maternal age" [3,4]. "In vitro fertilization may be considered as one of the reasons for increase in incidences of placenta percreta in modern scenario" [5,6]. Clark and colleagues studied "the relationship between previous CS and placental abnormalities and noted that risk of placental disorders including placenta previa increases with the number of previous cesarean sections" [7].

In literature, the first trimester placenta accreta cases have mostly been reported to occur after dilatation and curettage procedures. However, Horneman et al. [8] reported "a case of uterine rupture associated with placenta accreta developing in the second trimester. The common factor in first and early second trimester placenta previa cases is the history of previous CS and/or uterine curettage like in our case". In nations other than the United States, such as Japan, Turkey, Mexico, and Germany, a few cases of spontaneous uterine rupture due to placenta percreta have been described. All of these women had hysterectomy. This fatal complication of early percreta develops when the myometrium thins due to invasion of the placental villi into the myometrium at the site of placental implantation (especially at the previous scar site), causing the uterus to rupture. In our case too, we predicted a similar outcome considering the thinned out myometrium on imaging and decided for direct hysterectomy in place of conservative management. Höpker et al. from Germany reported "a case presentation at 10 weeks gestation in a patient who was suspected to have molar pregnancy based on USG,CT and MRI as part of the work-up, all of which showed trophoblastic infiltration through the myometrium into the serosa" [9]. A D and C was undertaken for suspected molar, which resulted in severe hemorrhage, and the patient needed hysterectomy despite uterine artery ligation. Pathology revealed placenta percreta, without any evidence of hydatiform mole. This is very similar to our case,however she was directly posted for hysterectomy hence avoiding bleeding due to potential haemorrhage from a direct dilatation and curettage. Also, Pont et al. from France noted "a case of acute abdomen and hemoperitoneum at 13 weeks gestation" [10]. The placenta percreta was discovered during a laparotomy, and the patient underwent a hysterectomy. All of these cases include undiagnosed placenta percreta in the first or second trimester, which resulted in significant blood loss, lengthy surgeries, and significant maternal morbidity. "Placenta percreta can be managed in two ways, first is surgical removal of the uterus and the surrounding tissues and another is conservative therapy including localized resection of the placental implantation site, oversewing, blunt dissection and packing techniques. The choice between hysterectomy and conservative therapy is dependent on the severity of the placenta percreta and associated complications" [11]. In our case, patient presented 3 months post her abortion with anemia which made her diagnosis even tricky.Although the possibility of molar pregnancy was considered due to late presentation post abortion ,her prior history of prev 2 lscs, low lying placenta in present pregnancy on early scan, incomplete curettage record, clinical and lab findings of S. BHCG levels and MRI findings gave us important clues to a more likely possibility of adherent placenta . She was counselled in detail about her condition and hysterectomy seemed to be the best option for her considering continued bleeding for 3 months, possibility of uterine rupture in future and her completed family history. Our elaborate planning in terms of correction of anemia pre-operatively and intra operatively, adequate anesthesia and OT back up, experienced gynaec oncologist to perform the surgery along with detailed counselling of patient and attenders ensured a smooth intra operative and post operative period without any serious maternal morbidity.

4. CONCLUSION

Placenta percreta can a silent killer if not detected on time and one should manage such cases in a tertiary care hospital preferably. Anticipation, preparation and action are the key factors involved in successful management of such high-risk patients.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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