Original Research Article

A Retrospective Study of Placenta cretas: A 4 year experience at Modern Government Maternity Hospital, Hyderabad

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Abstract

Background: Placenta accreta is a severe pregnancy complication and is currently the most common indication for peri partum hysterectomy. It is becoming an increasingly common complication mainly due to the increasing rate of cesarean delivery. Placenta accreta is considered a severe pregnancy complication that may be associated with massive and potentially life-threatening intrapartum and postpartum hemorrhage. It has become the leading cause of emergency hysterectomy. Maternal morbidity had been reported to occur in up to 60% and mortality in up to 7% of women with placenta accreta. In addition, the incidence of perinatal complications is also increased mainly due to preterm birth and small for gestational age fetuses. Placenta cretas are defined as abnormal adherences and/or ingrowths of the placenta to the uterine wall. Placenta creta is currently classified according to the depth of abnormal adhesion and invasion of the chorionic villi to the myometrium in the absence/deficiency of decidualization. The incidence of placenta accreta, defined as the abnormal adherence of the placenta to the uterine wall, has been increasing alarmingly in the developed as well as the developing world.. The exact pathogenesis of placenta accreta is unknown. Generally, placenta

accreta has been diagnosed on hysterectomy specimens when an area of accretion showed chorionic villi in direct contact with the myometrium and an absence of decidua or in placental basal plate.

Aim: The aim of this study was to determine the incidence of placentas cretas in our hospital and to profile the associated risk factors.

Materials and Methods: The patients who underwent gravid hysterectomies for placenta creta at Modern Government Maternity Hospital from 2013 to 2016 were included in study. A total of 25 cases during 4 year period were included out of 33063 deliveries. The indications for hysterectomy in majority of cases were heavy bleeding after removal of placenta or inability to remove the placenta manually either partially or totally. The specimens received in pathology department were thoroughly examined grossly and the representative sections were taken from all the specimens after proper fixation which was submitted for tissue processing and H&E stained sections were studied for final diagnosis.

Results: Amongst 33093 deliveries which occurred at our institute from January 2013 to December 2016, there were a total of 25 patients with placenta accreta diagnosed by histopathology, which was a rate of 0.7% cases per 1,000 . This total included 2 cases of focal placenta accreta (8%), 10 cases of placenta accreta vera (40%), 9 cases of placenta increta (36%), and 4 cases of placenta percreta (16%).

Conclusion: The incidence is considerably higher in women with both a previous caesarean delivery and placenta praevia. Therefore it is important to have a high index of suspicion in such cases. Women with a placenta previa overlying a uterine scar should be evaluated for the potential diagnosis of placenta accreta and arrangements should be made for delivery accordingly to reduce maternal and fetal morbidity and mortality.

Key words

Placenta creta, Placenta accrete, Placenta increta, Placenta percreta.

Introduction

The incidence of placenta accrete has been increasing alarmingly in the developed as well as the developing world. Pathological diagnosis relies on the finding of placental villi in direct opposition to myometrium, either in hysterectomy specimens or in placental basal plate. Placenta cretas are defined as abnormal adherences and/or ingrowths of the placenta to the uterine wall. Placenta creta is currently classified according to the depth of abnormal adhesion and invasion of the chorionic villi to the myometrium in the absence/deficiency of decidualization. There are three types of placenta accreta: placenta accreta vera (more often termed just placenta accreta) denotes placental attachment directly onto the myometrium without intervening decidua basalis, increta indicates invasion into the myometrium, percreta denotes penetration through the myometrium into or beyond the serosa, which may include involvement of nearby organs, including bladder or bowel. Placenta accreta can also be categorized according to the surface area of the placenta that is abnormally adherent, as focal, partial or total.[(1)] Because of abnormal attachment to the myometrium, placenta accreta is associated with an increased risk of heavy bleeding at the time of attempted vaginal delivery [1-4]. The need for transfusion of blood products is frequent, and hysterectomy is sometimes required to control life-threatening hemorrhage.

The exact pathogenesis of placenta accreta is unknown. Trophoblastic stem cells proliferate tremendously in order to generate both villous and nonvillous trophoblast (NVT), which includes both interstitial NVT and endovascular NVT [5].

NVTs, which are derived from the basal plate

and tips of the anchoring villi, invade through decidua, uteroplacental vessels, and myometrium; thereby anchoring the placenta to the uterine wall [2-4]. A subtype of EVT, termed "Interstitial Trophoblast" (IT) invades into the innermost third (inner sheet) of the myometrium, penetrating just a few millimeters [4].

Endovascular NVT binds to vascular basement membranes, invades these vessels, transiently plugs vascular lumens, and transiently replaces the endothelial lining. Endovascular NVT permits physiologically transformed vessels to massively increase blood flow into the intervillous space [5]. This transformation is regarded as complete when the entire circumference has been thus affected. Incomplete remodeling is present when less than half of the hyalinized stromal tissue of the uterine wall is replaced by EVTs [1, 9]. Abnormal vascular remodeling is also associated with placenta creta [6].

A proposed hypothesis includes a mal development of decidua, excessive trophoblastic invasion, or a combination of both. Tseng and Chou hypothesized that the abnormal expression of growth, angiogenesis, and invasion-related factors in the trophoblast populations are the main factors responsible for the occurrence of placenta accrete [7].

According to one study, approximately 95% of women diagnosed with placenta accreta have identifiable risk factors [8]. The most common presentation of placenta accreta is a woman with one or more previous cesarean deliveries and current placenta previa. There is robust evidence that the risk of placenta accreta also increases significantly with repeated cesarean deliveries [9-11].

Advanced maternal age, with delay in childbearing, has also emerged as a risk factor in developed countries [12]. In developing countries, risk factors for placenta accreta are multigravidity and high parity. Accreta is now seen in 9.3% of women with placenta previa [9]. Other risk factors include advanced maternal age, multiparity, Asherman syndrome, leiomyomata, radiation exposure, uterine anomalies, hypertension and smoking [9, 12-15].

Materials and methods

This study was conducted at Modern Government Maternity Hospital from 2012 to 2016. Patients who underwent gravid hysterectomies for placenta creta were included in our study and their clinical history including obstetric history, indication for all previous cesarean deliveries, and maternal and perinatal care and outcomes for the accreta pregnancy. The indications for hysterectomy in majority of cases were heavy bleeding after removal of placenta or inability to remove the placenta manually either partially or totally. A total of 33093 deliveries in MGMH during 4 year period, accounting up to 0.75 cases per 1000 deliveries. All the caesarian hysterectomy specimens were fixed in 10% formaline. After fixation thorough gross examination was done and representative tissue is submitted for processing and staining.

Results

Amongst 33093 deliveries which occurred at our institute from January 2013 to December 2016, there were a total of 25 patients with placenta accreta diagnosed by histopathology, which is a rate of 0.7% cases per 1,000. This total includes 2 cases of focal placenta accreta (8%), 10 cases of placenta accreta vera (40%), 9 cases of placenta increta (36%), and 4 cases of placenta percreta (16%) as per **Table - 1**.

Table – 1: Incidence of various accretes.

Type of Accreta	No. of cases
Placenta accreta vera	12
Placenta increta	9
Placenta percreta	4
Total	25

Patients aged ranged from 28 to 36 years mean age being 32±4 yrs. The mean parity was G3±1. There were 4 cases of grand multipara. Gestational age ranged from 33 to 38 weeks.

There were 3 cases where the gestational age was below 30 weeks all of which had associated co morbidities resulting in intrauterine death. One case at 27 weeks had associated pre eclampsia. Another case at 24 weeks had ante partum hemorrhage with severe oligohydramnios.

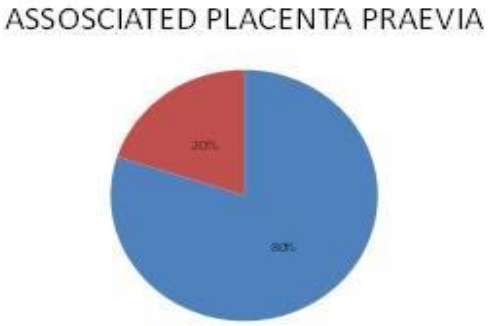
About 14 cases had history of 2 or more Lower segment caesarian section (LSCS), 8 cases had history of 1 LSCS and 3 cases had no H/o LSCS. History of prior uterine surgeries like myomectomy or D&C could not be obtained in these cases (**Table – 2**).

Table - 2: Association with CS.

History of caesarian section (CS)	%
NO CS	12%
1 CS	32%
2 or more CS	56%

History of Placenta praevia was present in 80% of cases. Most of the cases diagnosed with placenta previa had major previa (**Figure – 1**).

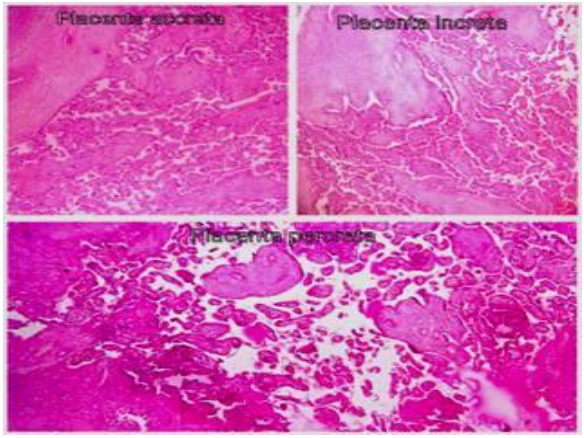
Figure – 1: Associated placenta previa.



Between types of accreta, there was no significant difference in average age or parity at accreta delivery. Maternal outcomes were significantly more severe among percreta cases in comparison with accreta vera. Foetal outcomes were favorable in most of the cases except intra uterine death (IUD) in 3 cases where associated comorbidities played a significant role. Gross morphology of placenta increta was as per **Figure – 2**. Microscopic image of various accretes were as per **Figure – 3**.

Figure - 2: Gross morphology of placenta increta.

Figure - 3: Microscopic image of various accretes.



Discussion

This study demonstrates the increasing incidence of placenta accreta/ increta/ percreta, the incidence being 0.7 per 1000 deliveries. The incidence of placenta accreta in various studies varies from 1 in 1600 to 1 in 12,000 patients [16-20].

In our study, placenta accreta vera was the commonest followed by placenta increta and percreta. There were only 2 cases of focal accreta probably due to conservative management where uterus was preserved. Our findings correlated with other studies where Placenta accreta constitute the majority of creta cases followed in decreasing frequency by placenta increta and placenta percreta [17, 18].

Placenta praevia [9, 12, 13] previous caesarean delivery [9, 12, 13], other previous uterine surgery [9], multiparity [9], advanced maternal

age [9, 12, 13] are factors that have previously been suggested as being associated with a higher risk of placenta accreta/ increta/ percreta. Our study investigated each of these factors and found that the risk of placenta accreta/ increta/ percreta was independently increased in women with a previous caesarean delivery and in women with placenta praevia diagnosed antenatally. No definite association with pre eclampsia could be made out as we had only 2 cases with pre eclampsia as associated co morbidity.

The risk of developing placenta accreta increases with the number of previous cesarean deliveries. Increasing numbers of previous caesarean section magnify this risk, with an eight-fold increase in the incidence of placenta accreta after two or more caesarean sections [12, 20]. Kastner, et al. found that the rates of placenta creta were 35, 56, 75, and 100% after zero, one, two, and more than two CSs, respectively [22]. In our smaller study the rates were 56%, 32% and 12% after 2 or more, 1 and no caesarean deliveries.

The presence of placenta previa is a well-recognized risk factor for placenta accrete [23]. In one study, up to 88% of the women have concomitant placenta previa [13]. The large prospective study of Silver, et al. found that the risk of placenta accreta was 0.03% in a patient undergoing her first CS if placenta previa was absent if placenta previa was present, the risk of creta was 3.3% at the first CS, but was 40% after the third CS [24]. In our study, 80% of cases had associated placenta praevia.

History of curettage and grand multiparity are also quoted in literature as other important risk factors. Our study could not evaluate these factors as history of curettage could not be obtained in most of the cases. Grand multipara constituted only 16% of our cases.

Conclusion

The incidence is considerably higher in women with both a previous caesarean delivery and placenta praevia. Therfore it is important to have a high index of suspicion in such cases. Women with a placenta previa overlying a uterine scar should be evaluated for the potential diagnosis of placenta accreta and arrangements should be made for delivery accordingly to reduce maternal and fetal morbidity and mortality.

References

1. Fox H, Sebire N. Pathology of the Placenta. 3rd edition. Elsevier Limited; 2007; p. 1-16, 80- 84.
2. Moser G, Gauster M, Orendi K, Glasner A, Theuerkauf R, Huppertz B. Endoglandular trophoblast, an alternative route of trophoblast invasion? Analysis with novel confrontation co-culture models. Hum Reprod., 2010; 25(5): 1127-369.
3. Naicker T, Khedun SM, Moodley J, Pijnenbo R. Quantitative analysis of trophoblast invasion in preeclampsia. Acta Obstet Gynecol Scand., 2003; 82(8): 722-9.
4. Khong TY, Robertson WB. Placenta creta and placenta praevia creta. Placenta, 1987; 8(4): 399-409.
5. Robertson WB. Uteroplacental vasculature. J Clin Pathol., 1976; 29(Suppl 1): 107–123.
6. Tantbirojn P, Crum CP, Parast MM. Pathophysiology of placenta creta: the role of decidua and extravillous trophoblast. Placenta, 2008; 29(7): 639-45.
7. Tseng JJ, Chou MM. Differential expression of growth-, angiogenesis- and invasion-related factors in the development of placenta accreta. Taiwan J Obstet Gynecol., 2006; 45(2): 100-6.
8. Eller A, Porter T, Soisson P, Silver R. Optimal management strategies for placenta accreta. BJOG: An International Journal of Obstetrics & Gynaecology, 2009; 116(5): 648-65429.
9. Gielchinsky Y, Rojansky N, Fasouliotis SJ, Ezra Y. Placenta accreta--summary of 10 years: a survey of 310 cases. Placenta, 2002; 23(2-3).

10. Ananth CV, Smulian JC, Vintzileos AM. The association of placenta previa with history of cesarean delivery and abortion: a metaanalysis. American journal of obstetrics and gynecology, 1997; 177(5).
11. Makoha FW, Felimban HM, Fathuddien MA, Roomi F, Ghabra T. Multiple cesarean section morbidity. International journal of gynaecology and obstetrics: the official organ of the International Federation of Gynaecology and Obstetrics, 2004; 87(3).
12. Wu S, Kocherginsky M, Hibbard JU. Abnormal placentation: twenty-year analysis. Am J Obstet Gynecol., 2005; 192: 1458–61.
13. Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta previa placenta accreta. American journal of obstetrics and gynecology, 1997; 177(1).
14. Shellhaas CS, Gilbert S, Landon MB, et al. The frequency and complication rates of hysterectomy accompanying cesarean delivery. Obstetrics and gynecology, 2009; 114(2 Pt 1): 1-10.
15. Belfort MA. Placenta accreta. American journal of obstetrics and gynecology, 2010; 203(5).
16. Benzineb NF, Battikh O, Sfar R. Placenta accreta, ten cases. J Gynecol Obstet Biol Reprod., 1995; 24: 751.
17. Mahmood TA, Kok KP. A review of placenta accreta at Aberdeen Maternity Hospital, Scotland. Aust N Z J Obstet Gynaecol., 1990; 30: 108.
18. Makhseed M, el-Tomi N, Moussa MA: A retrospective analysis of pathological placental implantation: Site and penetration. Int J Gynaecol Obstet., 1994; 47: 127.
19. Rashbaum WK, Gates EJ, Jones J, et al. Placenta accreta encountered during dilation and evacuation in the second trimester. Obstet Gynecol., 1995; 85: 701.
20. Sfar E, Zine S, Chaar N, et al. Analysis of placenta accreta risk factors. Rev Fr Gynecol Obstet., 1994; 89: 202.
21. Silver RM, Landon MB, Rouse DJ, Leveno KJ, Spong CY, Thom EA, et al. Maternal morbidity associated with multiple repeat cesarean deliveries. Obstet Gynecol., 2006; 107: 1226–32.
22. Kastner ES, Figueroa R, Garry D, Maulik D. Emergency peripartum hysterectomy: experience at a community teaching hospital. Obstet Gynecol., 2002; 99(6): 971-5.
23. Clark SL, Koonings PP, Phelan JP: Placenta previa/accreta and prior cesarean section. Obstet Gynecol., 1985; 66: 89.
24. Silver RM, Landon MB, Rouse DJ, Leveno KJ, Spong CY, Thom EA, et al. Maternal morbidity associated with multiple repeat cesarean deliveries. Obstet Gynecol., 2006; 107(6): 1226-32.