International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Elhabashy A et al. Int J Reprod Contracept Obstet Gynecol. 2025 Apr;14(4):1029-1034
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20250838

Original Research Article

Prevalence of sonographically detected fetal congenital malformations among cases of placenta accrete: a retrospective study

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Received: 01 January 2025
Accepted: 01 March 2025

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ABSTRACT

Background: Placenta accreta spectrum (PAS) is well known cause of maternal morbidity and mortality. Its incidence had been increased obviously during the last two decades. Almost all literature about placenta accreta is about maternal complications and paucity about fetal ones. Objectives were to assess the prevalence of fetal anomalies detected sonographically in cases placenta accreta diagnosed at the third trimester.
Methods: Our study is a retrospective study that had carried on at one tertiary center (Habashy 4D scan center; Alexandria; Egypt). We collected cases that diagnosed as placenta accreta prenatally by ultrasound in the third trimester from January 2020 till December 2024. We had excluded twin, ICSI pregnancies and maternal age ≥37 years. After enrollment of these cases we had searched for the prevalence of major fetal anomalies recorded in their reports.
Results: We included 133 PAS cases in our study. Maternal age window was: 24-36 years. Gestational age range at their third trimester scanning was: 28-36 weeks. 2 out of the 133 fetuses (1.995%) were had major fetal anomaly which were; transposition of the great arteries (TGA) and bladder exstrophy (BE).
Conclusions: The prevalence of major fetal anomalies among cases of placenta accreta is 1.995% which is lower than that in non-accreta pregnancies. Further large-scale studies are needed to confirm our observation.

Keywords: Placenta accreta, Fetal anomalies, Third trimester ultrasound

INTRODUCTION

Placental attachment disorders (PAD) represent an abnormal firm attachment between the extra villous trophoblast and the myometrium.[1] Histopathological assessment of cases with abnormally invasive placenta (AIP) reveled; absence of the Nitabuch layer (the decidual layer) between the placenta and the myometrium with histological evidence of chorionic villous invasion into the myometrium.[2,3]

The international federation of gynecology and obstetrics (FIGO) had endorsed the term PAS to cover all types of abnormally adherent placenta from clinical and histological points of view.[4,5] This means that currently the terms PAD and AIP had been replaced by the term PAS. There were two causes for such new nomenclature. Firstly; clinical and/or pathological grading of PAS has no long-term impact of the life of the patient; unlike cancer staging. In addition; about half of pathologists fail to accurately report the exact extent of villous attachment or invasion after peripartum hysterectomy.[6]

Several sonographic signs had been reported for prenatal diagnosis of PAS. The most important of them are three; loss of the clear zone, placental lacunae (±feeder vessels) and bridging vessels (on color or power Doppler mapping). The presence of these three sonographic signs together in a case with placenta previa anterior with history of previous CS; makes the probability of being PAS almost 100%. Other sonographic signs of PAS include: myometrial thinning, bladder wall interruption, placental bulge, exophytic mass, sub-placental and/or uterovesical hypervascularity.[1,7]

PAS is well known cause of maternal morbidity and mortality due to the massive hemorrhage that could occur antepartum, intrapartum and postpartum. Its incidence had been increased obviously during the last two decades.[8] The main risk for placenta accreta is previous caesarean section (CS) that was increased globally. The higher the number of previous CS; the higher the risk of placenta accreta will be and the higher likelihood of peripartum hysterectomy.[9]

Few studies had showed an association of placenta previa and the increased risk of fetal malformation, although this finding is still a debatable issue.[10-12] In early stages of embryo development (<10 weeks of gestation); hypoxia is normal till spiral arteries remodeling occurs. This hypoxia is not always be harmful to cells, and sometimes are beneficial and protective. Data from mouse studies suggest that oxygen consumption by cells of the embryo is normally low in early pregnancy, and this is called a "quiet metabolism", to decrease the production of the potentially harmful reactive oxygen species (ROS) and to protect the embryo from the free radical-mediated teratogenesis.[13]

Fibrous tissue in the CS scar may lead to persistence of low oxygen concentration after the first stages of embryonic development. This hypoxia may lead to defective fetal organogenesis especially congenital heart defects.

So theoretically; abnormal placentation in PAS could lead to hypoxia at the implantation site due to fibrosis at the uterine scar or attacks of bleeding that may disrupt the organogenesis.[11,12]

Almost all literature about placenta accreta is about maternal complications including morbidity and mortality as well as the surgical techniques for conservative or radical management. Too few articles targeted the fetal and neonatal impacts in pregnancies complicated with PAS.[10]

In our study; we will address the risk of congenital fetal malformations (CFM) associated with PAS.

Objectives

Objectives were to assess the prevalence of fetal anomalies detected sonographically in cases placenta accreta diagnosed at the third trimester.

METHODS

Our study is a retrospective study that had carried on at one tertiary center (Habashy 4D Scan center; Alexandria; Egypt). We collected cases that diagnosed as placenta accreta prenatally by ultrasound in the third trimester from January 2020 till December 2024. We had excluded twin, ICSI pregnancies and maternal age ≥37 years. After enrollment of these cases we had searched for the prevalence of major fetal anomalies recorded in their reports.

The ultrasound equipment used in our study was Voluson S10 Expert BT22 ultrasound system (GE Healthcare, Zipf, Austria), with transvaginal probe (GE RIC 5-9 MHz) and transabdominal probe (GE C 2-9). We scanned our cases during the third trimester of pregnancy (i.e. ≥28 weeks). All cases were scanned by both transabdominal ultrasound (TAUS) and transvaginal ultrasound (TVUS).

We used the following four sonographic signs for enrolment of cases in our study; loss or interruption of the clear zone, loss or interruption of bladder wall (uterovesical interface interruption), placental lacunae (± feeder vessels) and bridging vessels (or rail sign; on color or power Doppler mapping). Cases who had placenta previa anterior diagnosed by TVUS in addition to the previously mentioned four sonographic signs (together) were included in our study. Table 1 and Figure 1 showed the description of the sonographic signs that we had used in our study. All the included 133 cases of PAS in our study were proved as such intraoperatively (clinically) and/or postoperatively (by histopathology of hysterectomy specimens).

Table 1: Description of sonographic signs used in our study to diagnose PAS.[1,7,14]

Sonographic sign	Description
Placenta previa	Placenta is located in the lower uterine segment and the distance between its leading edge and the internal os is ≤20 mm
Clear zone interruption	Irregularity or loss of hypoechoic area in myometrium underneath placental bed
Uterovesical interface interruption	Interruption or loss of bright bladder wall (echogenic line between serosa of the uterus and bladder lumen)
Lacunae	Irregular anechoic intra-placental spaces that give the placenta a moth-eaten appearance. If lacunae are numerous, large and irregular; they are called Finberg grade 3. Lacunae are often containing turbulent flow visible on the grayscale imaging
Lacunae feeder vessels	Vessels with high velocity blood flow originating from the deep arterial vasculature of the myometrium; i.e. arcuate or radial arteries, and feeding the lacunae. Sometimes called: "tornado blood flow".
Bridging vessels (rail sign)	Vessels appearing to extend from placenta across myometrium and beyond serosa into bladder and running perpendicular to myometrium

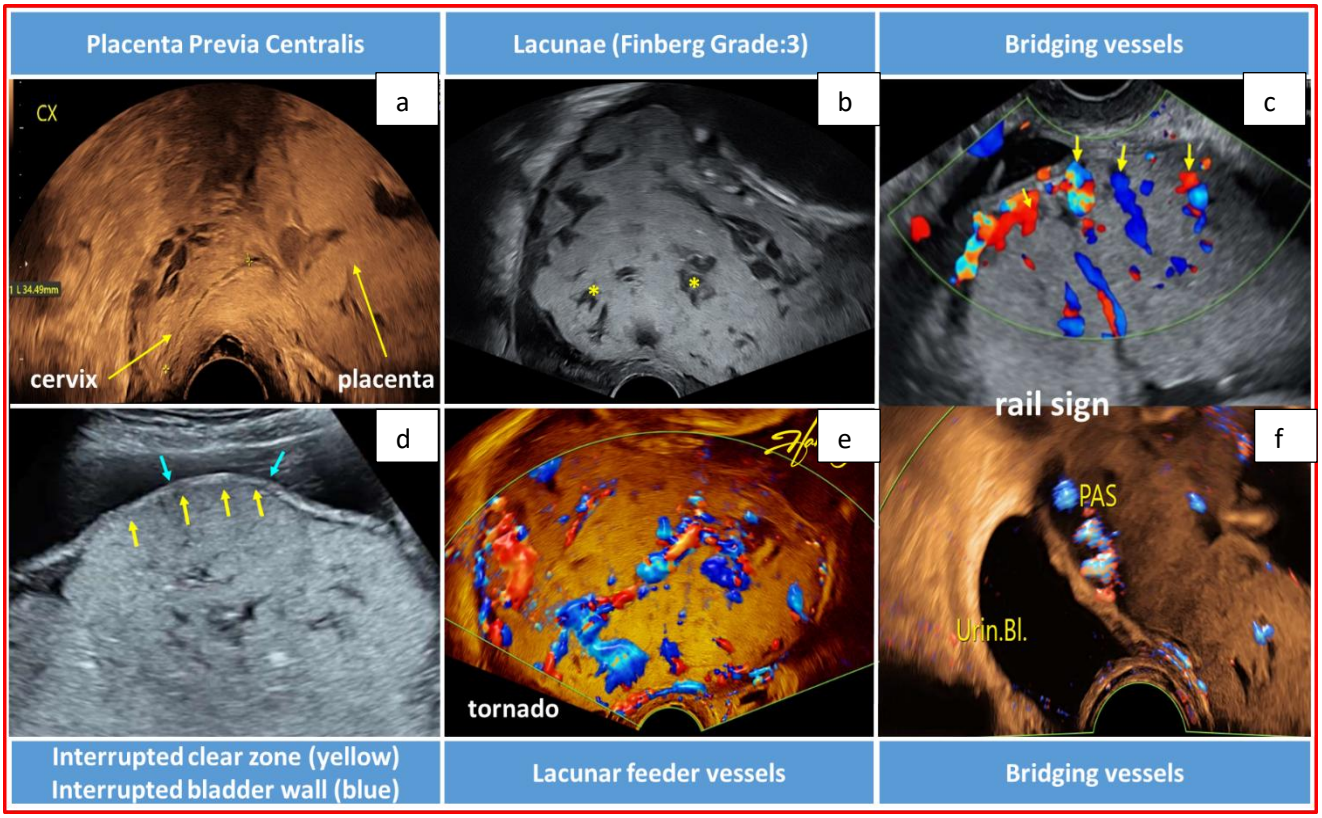

Figure 1 (a-f): Sonographic signs used in our study for diagnosis of PAS in the third trimester of pregnancy.

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). Categorical data were represented as numbers and percentages. Student t test was used to compare two groups for normally distributed quantitative variables.

RESULTS

We included 133 PAS cases in our study. Maternal age window was: 24-36 years. Gestational age range at their third trimester scanning was: 28-36 weeks. We had excluded three confounding factors for fetal anomalies; which are: twin, ICSI pregnancy and maternal age ≥37 years.

Table 2: Demographic data of all cases in our study.

Variables	Total number of PAS cases, 133
Age (range); (in years)	24-36
Parity (mean±SD)	3±2
Previous CS	133 (100%)
Previous vaginal birth	22 cases (16.5%)
Previous D and C	11 cases (8.27%)
Consanguinity	45 cases (33.83%)
Maternal medical disorders	16 (12.03%)
Maternal teratogen intake	7 (5.26%)
Previous anomalous sibling	3 (2.26%)

Table 2 showed the demographic data of our cases. Table 3 showed comparison of the demographic data in PAS cases diagnosed with fetal anomalies with those who were not have anomalous fetuses. There were no statistically significant differences between cases with anomaly and those without anomaly as regard: maternal age, gestational age at diagnosis and gestational age at delivery. The presence of risks for fetal anomalies was significantly higher in case who diagnosed with congenital fetal anomalies than in those who did not have anomalous fetus.

Table 3: Comparison of demographic data between cases with anomaly and cases without anomaly.

Variables	No anomaly, (n=12)	Fetal anomaly, (n=1688)	P value
Age (in years), mean±SD	30±6	29±7	0.804
Parity, mean±SD	4±1	3±2	0.976
GA at diagnosis (weeks), mean±SD	31±2	32±1	0.923
GA at delivery (weeks), mean±SD	36±1	35±2	0.763
Risk of fetal anomaly	25 cases (19.08%)	1 case (50%)	0.001

Two out of the 133 fetuses (1.995%) were had major fetal anomaly which were; TGA and BE. Figure 2 showed the sonographic features of the two cases with CFM reported

in our study. As regard the case of ectopia vesicae (the left side of Figure: 2); there were persistently absent urinary bladder with low cord insertion in the fetal abdomen that was associated with a lump below the cord insertion which represent the BE. As regard case of the TGA (the right side of Figure 2); there were parallel outflow tracts in the five-chamber view with early branching left ventricular outflow tract and two vessels seen in the three vessels tracheal view (instead of the normal three vessels) which is known as the "i" sign.

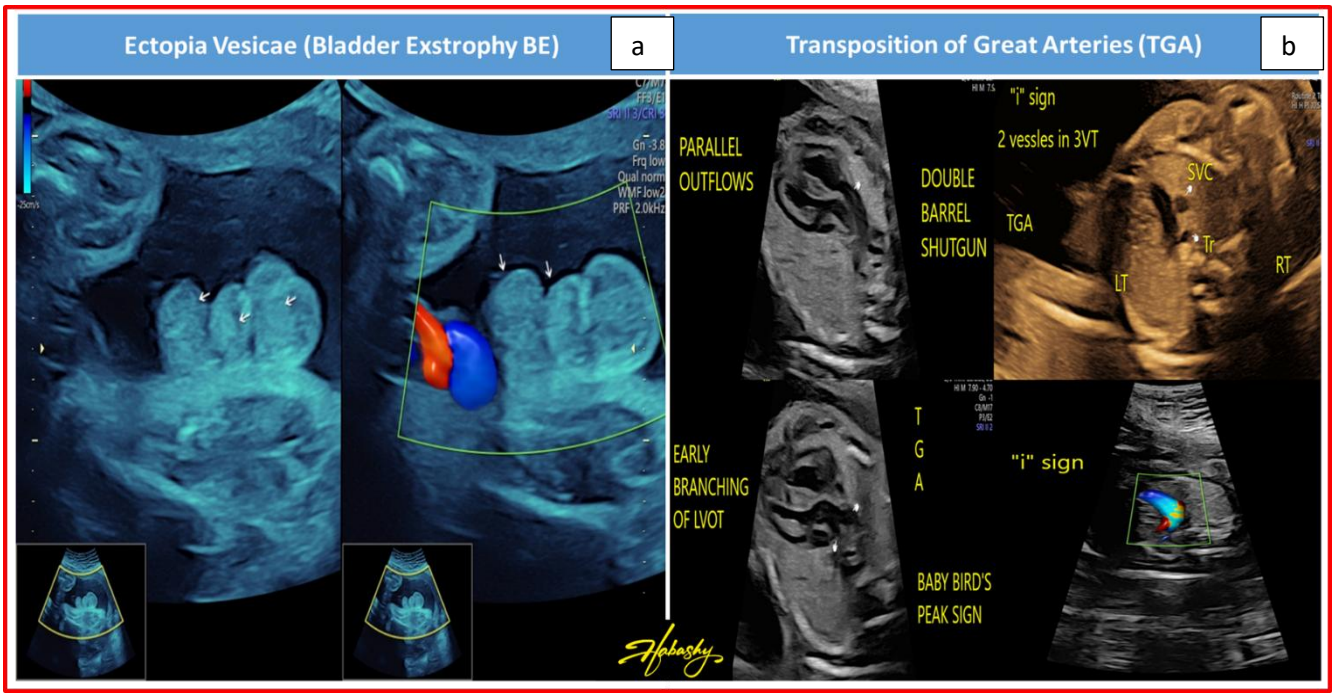

Figure 2 (a and b): Sonographic features of the two fetal anomalies that were detected in our cases.

DISCUSSION

PAS is well known cause of maternal morbidity and mortality. Its incidence had been increased obviously during the last two decades. Almost all literature about placenta accreta is about maternal complications and paucity about fetal ones. The aim of our study was to assess the prevalence of fetal anomalies detected sonographically in cases placenta accreta diagnosed at the third trimester.

Our study is a retrospective study that had carried on at one tertiary center (Habashy 4D scan center; Alexandria; Egypt). We collected cases that diagnosed as placenta accreta prenatally by ultrasound in the third trimester from January 2020 till December 2024. We had excluded twin, ICSI pregnancies and maternal age ≥37 years. After enrollment of these cases we had searched for the prevalence of major fetal anomalies recorded in their reports.

We included 133 PAS cases in our study. Maternal age window was: 24-36 years. Gestational age range at their third trimester scanning was: 28-36 weeks. Two out of the 133 fetuses (1.995%) were had major fetal anomaly which were; TGA and BE. Our results reveled a slightly higher malformation rate than the expected 2.58% (in general population) that reported by EUROCAT (European network of population-based registries for the epidemiologic surveillance of congenital anomalies).[15]

Pinto et al studied prospectively the fetal anomalies and neonatal complications among 311 pregnancies complicated by placenta accrete spectrum disorders.[10] The prevalence of fetal major congenital malformations in their study was 4.64% (15/323 newborns). The prevalence of fetal major congenital malformations among our cases was lower (1.995%); and this could be explained by two reasons. Firstly; number of cases in their study was higher than ours (311 VS 133 cases). In addition; we had excluded twin, ICSI pregnancies and maternal age ≥37 years; as these three factors are confounders for incidence of fetal anomalies. Viana Pinto et al group had not excluded twin and ICSI pregnancies from their study population. They also include cases up to maternal age of 48 years.

Kancherla et al studied the prevalence of fetal major congenital malformations among 1644 case affected by placenta previa over 10 years.[11] They founded that placenta previa was a risk of major fetal congenital malformations, after controlling for maternal age, parity, assisted conception and maternal medical disorders. 6.2% of their

study population had delivered infant with major congenital malformation.

The prevalence of fetal major congenital malformations among our cases was lower (1.995%); and this could be explained by two reasons.

Firstly; number of cases in their study was higher than ours (1644 vs 133 cases). In addition; they included placenta previa cases in general not placenta accrete spectrum in particular.

We have two limitations in our study. Firstly; we included only cases diagnosed at the third trimester of pregnancy so we had missed cases of placenta accreta diagnosed before the third trimester who opted termination of pregnancy to avoid maternal complications and/or due to associated fetal anomalies. In addition; we included fetal anomalies diagnosed prenatally in the third trimester and did not follow the cases postnatally; as about 5-11% of fetal congenital malformations missed prenatally.[16-18]

CONCLUSION

The prevalence of major fetal anomalies among cases of placenta accreta is 1.995% which is lower than that in non-accreta pregnancies. The risk of fetal anomalies as a consequence of placenta accrete is still inconclusive. Further large-scale studies are needed to confirm our observation.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Elhabashy A, El-Said R. Prevalence of sonographically detected fetal congenital malformations among cases of placenta accrete: a retrospective study. Int J Reprod Contracept Obstet Gynecol 2025;14:1029-34.