Review

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Placenta previa and the risk of intrauterine growth restriction (IUGR): a systematic review and meta-analysis

Abstract

Background: Whether placental location confers specific neonatal risks is controversial. In particular, whether placenta previa is associated with intra-uterine growth restriction (IUGR)/small for gestational age (SGA) remains a matter of debate.
Methods: We searched Medline, EMBASE, Google Scholar, Scopus, ISI Web of Science and Cochrane database search, as well as PubMed (www.pubmed.gov) until the end of December 2018 to conduct a systematic review and meta-analysis to determine the risk of IUGR/SGA in cases of placenta previa. We defined IUGR/SGA as birth weight below the 10th percentile, regardless of the terminology used in individual studies. Risk of bias was assessed using the Cochrane Handbook for Systematic Reviews of Interventions. We used odds ratios (OR) and a fixed effects (FE) model to calculate weighted estimates in a forest plot. Statistical homogeneity was checked with the I^2 statistic using Review Manager 5.3.5 (The Cochrane Collaboration, 2014).
Results: We obtained 357 records, of which 13 met the inclusion criteria. All study designs were retrospective in nature, and included 11 cohort and two case-control studies. A total of 1,593,226 singleton pregnancies were included, of which 10,575 had a placenta previa. The incidence of growth abnormalities was 8.7/100 births in cases of placenta previa vs. 5.8/100 births among controls. Relative to cases with alternative placental location, pregnancies with placenta previa were associated with a mild increase in the risk of IUGR/SGA, with a pooled OR [95% confidence interval (CI)] of 1.19 (1.10–1.27). Statistical heterogeneity was high with an $I^2 = 94\%$.
Conclusion: Neonates from pregnancies with placenta previa have a mild increase in the risk of IUGR/SGA.

Keywords: fetal growth restriction; intrauterine growth restriction; neonatal outcomes; placenta previa; small for gestational age.

Précis

Neonates from pregnancies with placenta previa have a mild increase in the risk of intra-uterine growth restriction (IUGR)/small for gestational age (SGA).

Introduction

Placenta previa refers to the presence of placental tissue that extends over the internal cervical os during pregnancy [1]. The incidence of this condition is reported to be 2% at 20 weeks of gestation, and through the process of placental migration known as trophotropism, decreases to around 4–6 per 1000 births between 34 and 39 weeks [2]. The primary risk factors for the development of placenta previa include a prior history of placenta previa, previous cesarean delivery, multiple gestations, use of fertility treatments and increasing maternal age, among others [3]. The risk of recurrence in subsequent pregnancies is reported at 4–8% [4].

Given its location over the cervical os, a proportion of the placental surface is exposed and lacks a proper uteroplacental interphase. Well-established sequelae of this condition include the potential for severe antenatal bleeding and preterm birth, as well as the need for cesarean delivery [3, 5]. The risk of bleeding is thought to occur when uterine contractions or gradual changes in the cervix and lower uterine segment apply shearing forces to the inelastic placental attachment site, resulting in partial detachment.

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For this reason, it is theorized that a lower placental-decidua surface area may confer an increased risk of adverse neonatal outcomes, including neonatal anemia, respiratory distress, hyperbilirubinemia, neonatal intensive care unit (NICU) admission and perinatal death [1]. Though given its pathophysiology of decreased placental surface and inadequate circulation may in theory explain an increased risk of adverse neonatal outcomes, whether the risk of intrauterine growth restriction (IUGR)/small for gestational age (SGA) is increased in placenta previa remains a matter of controversy [6, 7]. In this study, we conducted a systematic review and meta-analysis to determine the risk of IUGR/SGA in pregnancies complicated by placenta previa relative to those with normal placental insertion sites.

Materials and methods

Sources

We performed a Medline, EMBASE, Google Scholar, Scopus, ISI Web of Science and Cochrane database search, as well as PubMed (www.pubmed.gov) search until the end of December 2018 from the last 30 years using the following Boolean search criteria: "(Placenta previa OR previa) AND (intrauterine growth restriction OR IUGR) OR (fetal growth restriction OR FGR) OR (small for gestational age OR SGA) OR (low birth weight OR LBW)". Other than restricting the search to human studies and to articles in English, other limiting categorical terms used were the restriction to studies with singleton pregnancies and without any evidence of aneuploidy, congenital anomalies or placental trophoblastic invasion (accreta spectrum). The reference lists and bibliographies of included studies were then searched for other salient and pertinent manuscripts. Finally, manual searches of studies belonging to research teams having prior publications on placenta previa and growth restriction were undertaken, and other pertinent studies retrieved. This review was modeled on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement, and search flowchart depicting the search strategy is illustrated in Figure 1.
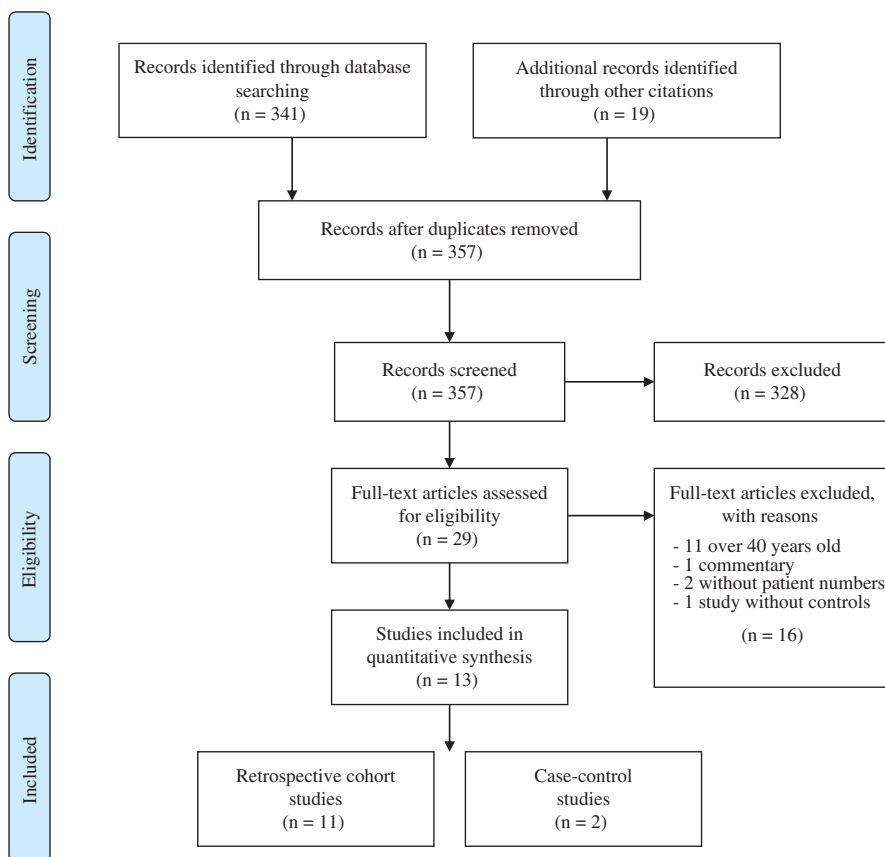

Figure 1: PRISMA flowchart for the systematic review of studies.

Study selection

All authors independently examined the electronic search results for reports of possibly relevant studies and those reports were retrieved and analyzed in further detail. Published studies were eligible for inclusion if they compared the risk of IUGR/SGA between cases of placenta previa and cases of normal placental location. In undertaking our search, we defined IUGR/SGA as birth weight below the 10th percentile, regardless of the terminology used in individual studies. Indeed, we included studies, which used terminology such as "small for gestational age", "fetal growth restriction" and "low birth weight", provided that they referred to neonates born under the 10th percentile corrected for gestational age. In addition, we made no distinction between the mode of diagnosis of placenta previa, the placental orientation, the presence of antepartum bleeding, nor the year or country of origin of the study in question.

All studies were assessed following predetermined quality criteria including the presence of a power calculation, the unit of analysis used and risk of bias. Risk of bias was assessed using the Cochrane Handbook for Systematic Reviews of Interventions (results not shown). We attempted to contact one author to obtain supplementary information regarding their study, but did not receive a response. As such, we did not include the study in question into our analysis for lack of complete information.

We used odds ratios (OR) and a fixed effects (FE) model with the Mantel-Haenszel method to calculate weighted estimates and their 95% confidence intervals (CI) where appropriate. A forest plot and a funnel plot are provided for visualization of the results (Figures 2 and 3). Statistical heterogeneity between results of studies was examined by inspecting the scatter in the data points on the graphs and the overlap of CIs, and by checking the χ^2 and I^2 statistics. The Review Manager 5.3.5 software (Version 5.3. Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014) was used to combine data for the meta-analysis. This meta-analysis was exempt from Institutional Review Board (IRB) approval because of the nature of the research design (review article), as well as the lack of use of identified patient data.

Results

We obtained 357 records, of which 13 met the inclusion criteria. All study designs were retrospective in nature, and included 11 cohort and two case-control studies. Study characteristics and individual inclusion criteria are provided in Table 1. A total of 1,593,226 singleton
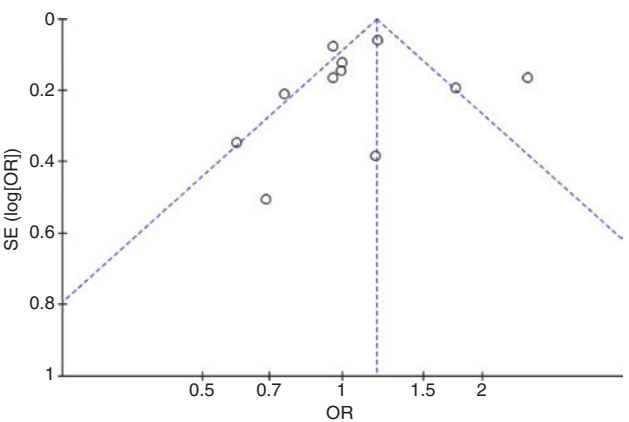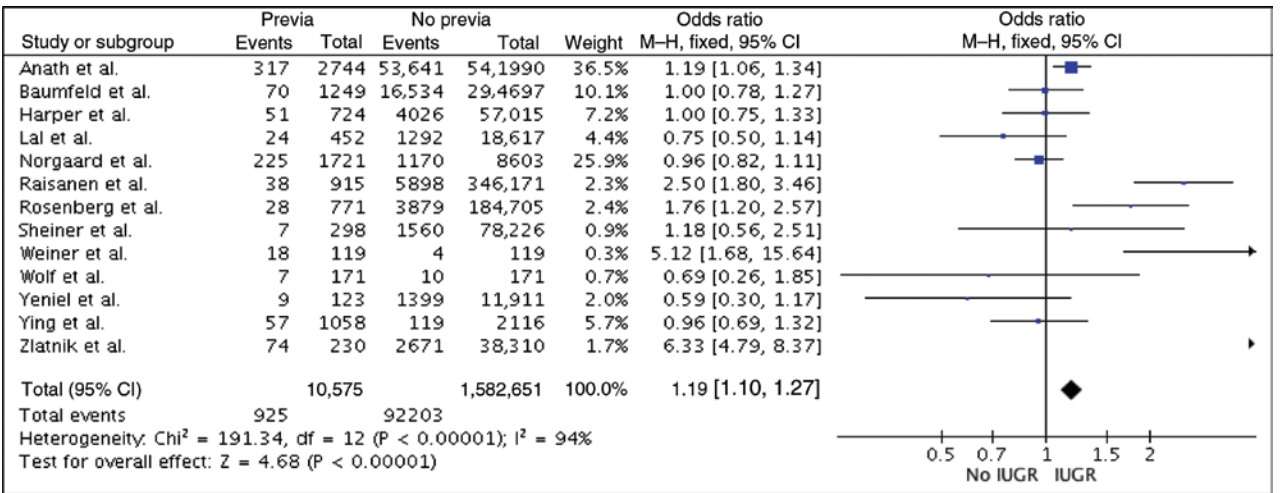

Figure 3: Funnel plot.

Figure 2: Forest plot – risk of IUGR/SGA in placenta previa.

Study or subgroup	Previa Events	Total	No previa Events	Total	Weight	Odds ratio M–H, fixed, 95% CI
Anath et al.	317	2744	53,641	54,1990	36.5%	1.19 [1.06, 1.34]
Baumfeld et al.	70	1249	16,534	29,4697	10.1%	1.00 [0.78, 1.27]
Harper et al.	51	724	4026	57,015	7.2%	1.00 [0.75, 1.33]
Lal et al.	24	452	1292	18,617	4.4%	0.75 [0.50, 1.14]
Norgaard et al.	225	1721	1170	8603	25.9%	0.96 [0.82, 1.11]
Raisanen et al.	38	915	5898	346,171	2.3%	2.50 [1.80, 3.46]
Rosenberg et al.	28	771	3879	184,705	2.4%	1.76 [1.20, 2.57]
Sheiner et al.	7	298	1560	78,226	0.9%	1.18 [0.56, 2.51]
Weiner et al.	18	119	4	119	0.3%	5.12 [1.68, 15.64]
Wolf et al.	7	171	10	171	0.7%	0.69 [0.26, 1.85]
Yeniel et al.	9	123	1399	11,911	2.0%	0.59 [0.30, 1.17]
Ying et al.	57	1058	119	2116	5.7%	0.96 [0.69, 1.32]
Zlatnik et al.	74	230	2671	38,310	1.7%	6.33 [4.79, 8.37]
Total (95% CI)		10,575		1,582,651	100.0%	1.19 [1.10, 1.27]
Total events	925		92203			

Heterogeneity: Chi² = 191.34, df = 12 (P < 0.00001); I² = 94%
Test for overall effect: Z = 4.68 (P < 0.00001)

Table 1: Study characteristics.

	Year	Country	Design	n	Inclusion criteria
Ananth et al. [7]	2001	New Jersey, USA	Retrospective cohort study	544,734 (previa = 2744)	– Singleton live delivery in NJ hospital between 1989 and 1993 – Restricted to cases of previa delivered by c/s – SGA defined as <10th%
Baumfeld et al. [8]	2016	Beer Sheva, Israel	Retrospective cohort study	295,946 (previa = 1249)	– Deliveries 1998–2013 at Soroka University Medical Center with pregnancy complicated by placenta previa – Excluded: multiple gestation and fetal chromosomal/congenital malformation – IUGR defined as growth <10th% Placenta previa dx sonographically
Harper et al. [6]	2010	Washington, USA	Retrospective cohort study	57,739 (previa = 724)	– Singleton pregnancy delivered after 20 weeks of gestation, at single tertiary center 1990–2008 – Excluded: fetal demise/major fetal anomalies/multiple gestation, marginal previa – Placenta previa diagnosis confirmed by TV US – IUGR defined as birth weight <10th% using Alexander growth standard for GA at delivery
Lal and Hibbard [9]	2015	USA	Retrospective cohort study	19,069 (previa = 452)	– First singleton pregnancy in Consortium on Safe Labor database with placenta previa noted at admission or as reason for c/s – SGA defined as <10th% birth weight
Nørgaard et al. [10]	2012	Denmark	Retrospective cohort study	10,324 (previa = 1721)	– Singleton deliveries with placenta previa during 2001–2006 – Excluded: delivery <22 weeks – Placenta previa confirmed in the third trimester by US/bleeding in the second trimester + placenta covering internal cervical os on US
Räisänen et al. [11]	2014	Finland	Retrospective cohort study	596,562 (previa = 1540)	– All singleton birth >22 weeks of GA (or >500 g) on Finnish Medical Birth Register and Welfare 2000–2010 – Excluded: major congenital anomalies/multiple gestation, birth missing information on GA, birth weight or parity – Placenta previa diagnosed on US, confirmed in the third trimester – SGA defined as BW <2 standard deviations below the mean (Finnish population-based birth curves)
Rosenberg et al. [3]	2010	Beer Sheva, Israel	Retrospective cohort study	185,476 (previa = 771)	– All singleton pregnancy with placenta previa, delivered at Soroka University Medical Center 1988–2009 – Excluded: Multiple fetuses/pregnancies w/o adequate prenatal surveillance Previa diagnosed by US in the second or third trimester
Sheiner et al. [12]	2001	Beer Sheva, Israel	Retrospective cohort study	78,524 (previa = 298)	– All computerized singleton deliveries at Soroka University Medical Center 1990–1998 Placenta previa defined as placental attachment totally/mostly in lower uterine segment, covering internal os in the third trimester, diagnosed by US and during labor
Weiner et al. [13]	2016	Holon, Israel	Case-control study	238 (previa = 119)	– Cesarean deliveries for placenta previa between 24 and 42 weeks of GA between 2009 and 2015 – Excluded: multiple gestation, deliveries <24 weeks, known fetal/neonatal malformation, cases with concurrent placenta accreta and cases missing data – Control = elective c/s during same period, for malpresentation or previous c/s and placenta sent to pathology Diagnosis of placenta previa confirmed in all women in the third trimester TV US + placenta sent to pathology

Table 1 (continued)

	Year	Country	Design	n	Inclusion criteria
Wolf et al. [14]	1991	Connecticut, USA	Case-control study	342 (previa = 171)	– Deliveries between 1980 and 1990 in affiliated hospitals of MFM division of University of Connecticut – Known gestational age (two of gestational age criteria) – Excluded: cases with chronic HTN, pregnancy induce HTN, multiple gestation, insulin dependent diabetes, fetal/neonatal anomalies – Placenta previa defined as implantation in the lower uterine segment in advance of the presenting part – Control = patient matched for race, parity, GA at delivery and fetal gender – SGA defined as BW <10th% of GA
Yeniel et al. [15]	2012	Turkey	Retrospective cohort study	12,034 (previa = 123)	– Singleton gestation delivered between 20 and 42 weeks – Excluded: fetal anomaly cases, questionable placenta previa diagnosis – Placenta previa defined as implantation over cervical os. Determined by the second/third trimester US and at c/s – FGR defined as BW <10th% using Alexander growth monogram for GA at delivery
Ying et al. [16]	2016	Shanghai, China	Retrospective cohort study	3174 (previa = 1058)	– Singleton pregnancy with abnormal position of placenta delivered by c/section at a tertiary hospital, between 2010 and 2014 – Excluded: low lying placenta, history of gestational HTN/pre-eclampsia, abnormal gestation or birth history, recurrent spontaneous abortion, use of specific medications during pregnancy (ASA, LMWH, glucocorticoids), lethal fetal malformation, complication during pregnancy (chronic HTN, CKD, DMII, hyper/hypothyroidism, auto-immune disease) Placenta previa diagnosis confirmed by TV US between 32 and 36 weeks
Zlatnik et al. [17]	2007	California, USA	Retrospective cohort study	38,540 (previa = 230)	– All singleton delivery between 1980 and 2006 at Moffit-Long Hospital – Excluded: delivery <24 weeks GA, fetuses with known lethal congenital anomalies, multifetal gestation and maternal transports – Placenta previa diagnosed by US in the second trimester and confirmed subsequently

TV US, transvaginal ultrasound; GA, gestational age; c/s, cesarean section; BW, birth weight; HTN, hypertension; FGR, fetal growth restriction; ASA, acetyl salicylic acid; LMWH, low molecular weight heparin; CKD, chronic kidney disease; DMII, type II diabetes mellitus; MFM, Maternal-Fetal Medicine.

pregnancies were included, of which 10,575 had a placenta previa. The incidence of growth abnormalities was 8.7/100 births in cases of placenta previa vs. 5.8/100 births among controls. Relative to cases with alternative placental location, pregnancies with placenta previa were associated with a mild increase in the risk of IUGR/SGA, with a pooled OR (95% CI) of 1.19 (1.10–1.27) (forest plot – Figure 2). A funnel plot is provided in Figure 2 for visualization of the results. Statistical heterogeneity was high with an $I^2 = 94\%$.

Discussion

The most common presentation of placenta previa is as a finding on routine ultrasound examination at about 16–20 weeks of gestation for assessment of gestational age, fetal anatomic survey or prenatal diagnosis [1, 3]. The diagnosis of placenta previa requires the identification of echogenic homogeneous placental tissue over the internal cervical os [18]. One to six percent of pregnant women are found to have sonographic evidence

of a placenta previa on these examinations [19]. When placenta previa persists near term, several important adverse maternal and neonatal outcomes may ensue. However, whether placenta previa increases the risk of IUGR/SGA is controversial. In this meta-analysis of over 1,593,226 singleton pregnancies and 10,575 confirmed cases of placenta previa, we show that placenta previa increases the relative risk of IUGR/SGA by an average of 19% (95% CI 10–27%).

Whether one should consider placenta previa in the same category as other placental insertion abnormalities is unknown. Nevertheless, several clinical associations with other placental conditions exist, which may point to an underlying genetic susceptibility. One hypothesis is that the presence of areas of suboptimally vascularized decidua in the upper uterine cavity due to previous surgery or multiple pregnancies promotes implantation of trophoblast in healthy decidua near the lower uterine cavity [20].

It is well documented that when placenta previa is diagnosed, the possibility of placenta previa-accreta should be considered [21]. In a prospective study of women with placenta previa undergoing cesarean delivery, the frequency of placenta accreta increased proportionally with an increasing number of cesarean deliveries. Similarly, placenta previa is a known risk factor for vasa previa and velamentous umbilical cord insertion [19, 21].

Despite the controversial evidence, several theories have attempted to explain the potential etiologies of an association between placenta previa and growth abnormalities in the fetus. First, as the muscle mass and contractile work of the corpus uteri and fundus are greater than in the lower uterine segment, the blood supply to the latter is likely lower, presumably resulting in lower perfusion for a low-lying placenta previa. Similarly, repeated bleeding episodes from placenta previa may impact fetal oxygenation and growth. In keeping with this theory, numerous reports have documented an increased risk of fetal anemia in cases of placenta previa, which may be related both to intra-placental bleeding of fetal origin during pregnancy and to the incision through the fetal vessels in the placenta during cesarean delivery [1]. Finally, whether epidemiologic confounders or modifying variables in certain studies account for an increased risk of IUGR/SGA has been observed. The prime example is the study by Räisänen et al. [11], which finds an increased risk of IUGR with placenta previa, only among multiparous, but not nulliparous women. That said, differences in the risk of fetal growth restriction have been observed depending on whether univariate or multivariate analyses are undertaken [7, 20, 22, 23].

Clinical implications of the findings

The management of pregnancies at risk of IUGR includes the provision of prenatal screening, the adequate assessment of fetal anatomy, the continuous monitoring of fetal biometry throughout gestation and, in select cases, the provision of anticoagulation or anti-platelet therapy [24]. As with most normal pregnancies, before a placenta previa is diagnosed, the vast majority of patients will have undergone regular prenatal screening and evaluation of fetal anatomy. Given the need to re-assess placental location and its distance to the internal os in the third trimester to plan for delivery, fetal growth is already likely to be estimated at that time again. Moreover, given the substantial risk of antepartum hemorrhage, whether the provision of aspirin and anticoagulation in women with placenta previa increases such risk has not been studied. Therefore, at the present time, the findings of this study are unlikely to alter the current clinical management of these pregnancies.

That said, once IUGR is established, fetal Doppler studies are recommended to screen for signs of placental insufficiency and fetal distress [24]. Depending on the etiology and severity of IUGR, delivery is likely to be induced around 38–39 weeks of gestation. However, given the risk of catastrophic hemorrhage, the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development and the Society for Maternal-Fetal Medicine recommend that pregnancies with uncomplicated placenta previa be delivered between 36 + 0 and 37 + 6 weeks of gestation [25]. Therefore, at the present time, there is no evidence that specifically monitoring fetal growth or Doppler studies with serial ultrasound examinations is useful in cases of placenta previa. Given the findings of this study, which only suggest a mild increase in the risk of IUGR/SGA, we do not propose that further screening with serial scans be undertaken at this time. Nevertheless, what the findings of this study may in fact prompt is a more comprehensive counseling to patients, as well as to the NICU team, who may care for the neonates with this condition, often born in the late-preterm or early-term period.

Strengths and limitations

The strengths of this study are multiple and include the vast number of patients analyzed, the homogeneity of the inclusion criteria across studies, as well as the use of proper statistical techniques. Similarly, to the best of our knowledge, ours is the first and only meta-analysis in the literature addressing the association between placenta

previa and growth restriction, an increasingly important and relevant question in clinical medicine today. On the other hand, the limitations of our study are several and worth mentioning. First, while all of the studies included in the meta-analysis addressed the outcome of IUGR, the definitions are not strictly identical and as such, they may introduce bias into our estimates. Indeed, the literature is notoriously heterogeneous with regard to terminology about growth restriction, with some reports considering growth under the 10th percentile as diagnostic while others do so in cases under the fifth or third centile. That said, all weight estimates were sonographically determined and the same criteria were used for all patients in each individual study. Likewise, though individual studies may use a different terminology, be it IUGR or SGA, all refer to fetuses whose growth falls below the 10th percentile for gestational age. Second, though the pooled number of patients is high, statistical heterogeneity between studies was large as well, with an I^2 of 94%. Third, given the retrospective nature of the study designs included in the meta-analysis, adjusting for different confounders in individual studies may have introduced information bias. To mitigate these, we conducted sensitivity analyses looking at univariate and multivariate estimates in individual studies, and our findings and conclusion remained unchanged. Finally, despite the robustness of our findings, we cannot prove a causal relationship between placenta previa and IUGR/SGA.

Conclusion

Neonates from pregnancies with placenta previa have a mild but significant increase in the risk of IUGR/SGA. A greater understanding of placenta previa and its consequences can allow providers to better inform, counsel and manage patients with this condition. Whether the degree of IUGR/SGA is significant and whether it requires increased surveillance and/or prompt delivery are outside the scope of the current study. Future studies should attempt to address this question.

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