Timing of Delivery and Neonatal Outcomes for Small-for-Gestational-Age Fetuses

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Article includes CME test

Objectives—To investigate whether antenatal recognition of small-for-gestational-age (SGA) fetuses with normal maternal and fetal Doppler values delivered after 34 weeks' gestation is associated with changes in the risk of adverse maternal and neonatal outcomes.

Methods—In this retrospective study, we included 313 singleton SGA fetuses and 313 appropriate-for-gestational-age control fetuses born between 34 and 42 weeks' gestation from 2009 to 2012. Small-for-gestational-age fetuses identified before delivery (n = 124), for whom antenatal surveillance was performed until delivery (estimated fetal weight twice weekly and Doppler evaluation of the fetal compartment once weekly), were compared to those not identified at delivery (n = 189). The latter group did not undergo antenatal surveillance for several reasons (women for whom a sonographic evaluation or gynecologic consultation was not performed in the third trimester and incorrect sonographic biometric evaluation in the third trimester). Main outcome measures were mode of delivery, perinatal complications, and neonatal intensive care unit admission. The risk of serious fetal complications was assessed by cross-tabulation analysis adjusted for gestational age and degree of SGA.

Results—Prenatally recognized SGA fetuses were smaller and delivered earlier than unrecognized SGA fetuses ($P < .05$). Fetal acidemia (pH <7.10) was significantly more common in unrecognized SGA fetuses (3.7% versus 0%). Small-for-gestational-age fetuses at or below the 3rd percentile were more commonly recognized prenatally and hospitalized in the neonatal intensive care unit. Unrecognized SGA fetuses also had worse fetal outcomes compared to controls ($P < .05$). Recognized and unrecognized SGA fetuses were born significantly more frequently by cesarean delivery ($P < .05$). No significant differences in perinatal outcomes were found between recognized SGA deliveries with or without medical induction.

Conclusions—Antenatal recognition of SGA fetuses delivered after 34 weeks' gestation might improve perinatal outcomes. Medical induction of labor did not modify neonatal outcomes among prenatally recognized SGA fetuses.

Key Words—intrauterine growth restriction; medical induction of labor; neonatal outcome; obstetric ultrasound; small-for-gestational-age neonate

Received October 16, 2013, from the Department of Woman's and Child's Health, Maternal-Fetal Medicine Unit, University of Padua School of Medicine, Padua, Italy (S.V., F.G., D.T., V.Z., G.A., E.C.); and Clinic of Obstetrics and Gynecology, Department of Experimental Clinical and Medical Sciences, University of Udine, Udine, Italy (A.P.L.). Revision requested November 15, 2013. Revised manuscript accepted for publication January 12, 2014.

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Abbreviations

AGA, appropriate-for-gestational-age; GA, gestational age; IUGR, intrauterine growth restriction; NICU, neonatal intensive care unit; PI, pulsatility index; SGA, small-for-gestational-age

doi:10.7863/ultra.33.10.1721

Intrauterine growth restriction (IUGR) complicates approximately 10% of all pregnancies, and around 80% of these fetuses are born at term.[1] There is still a debate in the literature about the definition of IUGR. The American College of Obstetricians and Gynecologists uses the terms IUGR and fetal growth restriction interchangeably to identify fetuses with sonographically estimated fetal weight below the 10th percentile for gestational age (GA),

whereas small-for-gestational-age (SGA) is used to identify neonates whose birth weight is below the 10th percentile for GA.[1] However, the Royal College of Obstetricians and Gynaecologists considers an abdominal circumference less then 10% sufficient to suspect an SGA fetus.[2]

Intrauterine growth-restricted fetuses with Doppler abnormalities are associated with a higher incidence of perinatal and neonatal morbidity, fetal distress during labor, and a higher cesarean delivery rate,[3–5] whereas SGA fetuses with normal umbilical artery Doppler values have more favorable outcomes. However, an impaired fetal outcome despite normal umbilical artery Doppler values at term is well described, and the general consensus is that the recognition of SGA involves an assessment of well-being and serial monitoring of growth until delivery.[1,6] Hitherto, fetal umbilical artery Doppler values have been used to distinguish IUGR due to placental insufficiency from constitutionally small fetuses, allowing a reduction in perinatal death, especially in preterm fetuses, although they lose this ability approaching term.[7] Recent studies suggest that a large proportion of SGA pregnancies have milder forms of late-onset IUGR with an increased risk of adverse perinatal outcomes, abnormal neonatal neurobehavioral performance, suboptimal neurodevelopment in childhood, and possible cardiovascular risk in adult life.[8–10] Identification of late IUGR actually is not based only on umbilical artery Doppler evaluation, which at term might be falsely reassuring, but also on cardiotocographic analysis and sonographic examination, including a biophysical profile and cerebroplacental ratio.[11] Small-for-gestational-age fetuses are often managed by labor induction, with an increased risk of cesarean delivery for a nonreassuring fetal status.[3] Predicting the risk might allow timing of delivery, assist in the decision-making process regarding labor induction, and result in more efficient provision of resources at delivery.[12,13]

The aim of this study was to investigate whether antenatal recognition of SGA fetuses delivered after 34 weeks' gestation with normal maternal and fetal Doppler values can reduce the risk of adverse maternal and neonatal morbidity and mortality with respect to unrecognized SGA fetuses.

Materials and Methods

Study Procedures

This retrospective study was performed between 2009 and 2012 in a tertiary referral center. All women with singleton cephalic pregnancies who delivered an SGA neonate at 34 weeks' gestation or later were eligible. Gestational age was calculated by considering the last menstrual period and determined by the crown-rump length during first-trimester sonography or fetal biometry during second-trimester sonography. The Ethics Committee and Institutional Review Board approved the study. We identified 313 SGA fetuses: 124 recognized prenatally and 189 unrecognized until after delivery. Recognized SGA fetuses were defined as having an estimated fetal weight below the 10th percentile, a fetal abdominal circumference below the 10th percentile, or flattening of the growth curve in the third trimester.[14,15] For those fetuses, antenatal surveillance was performed by fetal biometry every 2 weeks and maternal and fetal Doppler and amniotic fluid evaluations (1–3 times per week depending on severity) as established by hospital protocols. Fetal and maternal Doppler assessments were performed as previously described, considering the umbilical artery, middle cerebral artery, ductus venosus, and uterine arteries.[16] An umbilical artery pulsatility index (PI) higher than 2 SDs, a middle cerebral artery PI below the 5th percentile, and a ductus venosus PI above the 95th percentile were considered abnormal, as well as a cerebroplacental ratio below the 5th percentile.[17] An average uterine artery PI above the 95th percentile after 25 weeks' gestation was considered a sign of placental insufficiency.[18] When Doppler abnormalities were detected, the fetus was considered IUGR and was excluded from this group.

Unrecognized SGA fetuses were those who did not receive routine examinations for the following conditions: women without a third-trimester sonographic or gynecologic evaluation for economic reasons or for lack of consultation by a family physician and an incorrect evaluation during the third trimester. In these cases, the diagnosis of SGA fetuses was made at birth by neonatal weight below the 10th percentile with respect to published normograms,[15] and Doppler sonography was available at least 48 hours before delivery because of an internal hospital protocol that requires the control of fetal well-being by umbilical artery Doppler and amniotic fluid evaluations of every patient admitted for delivery. Furthermore, we randomly selected 313 appropriate-for-gestational-age (AGA) fetuses delivered at 34 weeks' gestation or later with normal fetal and maternal Doppler waveforms as controls.[19] All SGA fetuses included had normal umbilical artery and maternal Doppler waveforms.[19] Exclusion criteria were congenital and chromosomal defects, infections and maternal diseases such as diabetes mellitus and gestational diabetes, metabolic disorders, breech presentation, and hypertension during pregnancy.

In SGA fetuses, we adopted expectant management depending on the Doppler evaluation, cardiotocography, and fetal biophysical profile as previously defined.[16] Computerized cardiotocography for fetal surveillance was performed weekly from 34 to 36 weeks' gestation until delivery.

Fetal bradycardia or tachycardia, major deceleration, and a short-term variation less than 3.75 milliseconds were considered pathologic. In the absence of pathologic findings, the SGA pregnancies were followed until 40 weeks' gestation. Indications for delivery before 40 weeks in the recognized SGA group were fetal growth arrest, a pathologic fetal biophysical profile, cardiotocographic abnormalities (short-term variation <5 milliseconds, presence of bradycardia, tachycardia, or deceleration, and absence of variability), oligohydramnios (maximal amniotic fluid pocket <3 cm), and maternal indications (eg, gestational hypertension and preeclampsia). In the absence of spontaneous labor, medical induction was planned at 40 weeks' gestation. During labor, recognized SGA fetuses were monitored by continuous cardiotocography throughout delivery, whereas fetuses considered uncomplicated were monitored intermittently.

Information about the recognized and unrecognized SGA fetuses and AGA fetuses was gathered from labor room registry and clinical files. Baseline maternal characteristics were registered, and the following neonatal characteristics were reported: GA at delivery, mode of delivery, birth weight, Apgar score, metabolic status, and neonatal complications. In addition, we considered the following neonatal outcomes: acidemia (pH <7.15, <7.10, and <7.00), 5-minute Apgar score less than 7, bradypnea (<35 breaths per minute), symptomatic hypoglycemia (blood glucose <40 mg/dL with clinical signs), death before discharge, respiratory distress syndrome, anemia, jaundice, sepsis, intraventricular hemorrhage, periventricular leukomalacia, convulsions, cerebral palsy, meconium aspiration (respiratory distress caused by aspiration of meconium with supportive radiographic findings), necrotizing enterocolitis (defined according to the Bell criteria), and stillbirth. Secondary outcomes included cesarean delivery, instrumental vaginal delivery, neonatal intensive care unit (NICU) admission, and NICU hospitalization length.

Statistical Analysis

Categorical variables are presented as percent (number). Parametric distribution was assessed by the Kolmogorov-Smirnov test. Continuous variables are presented as mean ± standard deviation and (range) or median (interquartile range). Univariate analysis for categorical variables was performed by a χ^2 or Fisher exact test. For continuous variables, the following tests were used: independent-samples *t* test, 1-way analysis of variance, Wilcoxon test, and Kruskal-Wallis test. Multivariate logistic regression analysis was also performed. Data were analyzed by R version 2.15.2 software (www.r-project.org), and $P < .05$ was considered significant.

Results

The mean maternal age was 31.68 ± 5.29 years; the mean GA at delivery was 39.32 ± 1.43 weeks; and the proportion of multiparous women was 40.7% (255 of 626). Ethnicities of the study group were 83.2% white, 8.6% African-Caribbean, and 8.2% from other ethnicities. During the study period, only 39.6% (124) of SGA fetuses were recognized prenatally. All prenatally recognized SGA fetuses had normal Doppler values (Table 1), and we did not observe any intrauterine fetal deaths.

In Table 2, the differences between prenatally recognized and unrecognized SGA fetuses are shown. The SGA fetuses recognized prenatally were significantly smaller and delivered more commonly between 34 and 36 weeks' gestation than the unrecognized SGA fetuses ($P < .05$), whereas in the 37- to 38-week group, there was no difference, and in the 39- to 42-week group, there was a significantly higher prevalence of unrecognized SGA fetuses ($P < .05$). Fetuses with birth weight at or below the 3rd percentile were more common in the prenatally recognized than the unrecognized group ($P < .05$). Moreover, SGA fetuses recognized prenatally had a significantly lower prevalence of fetal acidemia (pH <7.10 or <7.15; $P < .05$). Furthermore, in the multivariate logistic regression analysis, prenatal SGA recognition significantly predicted a lower prevalence of pH lower than 7.10 (odds ratio, 0.06; 95% confidence interval, 0.01–0.78) and pH lower than 7.15 (odds ratio, 0.13; 95% confidence interval, 0.03–0.62; correction for maternal age, GA at delivery, labor induction, mode of delivery, amniotic fluid staining, and abnormal fetal heart rate). As shown in Table 3, no significant differences between recognized and unrecognized SGA fetuses were found for the reasons for NICU admission and neonatal complications during NICU hospitalization. In Table 4, we stratified the main fetal outcomes by GA at delivery, and we found a difference in the prevalence of

Table 1. Doppler Values in the Prenatally Recognized SGA Fetuses at the Last Sonographic Examination Before Delivery

Parameter	Value
Umbilical artery PI	0.92 ± 0.14 (0.52–1.35)
Middle cerebral artery PI	1.93 ± 0.23 (1.54–2.33)
Cerebroplacental ratio	2.20 ± 0.98 (1.14–4.40)
Ductus venosus PI	0.54 ± 0.11 (0.39–0.81)
Uterine artery PI	0.62 ± 0.10 (0.31–0.74)
Uterine artery RI	0.46 ± 0.08 (0.33–0.54)
Uterine artery bilateral notch	0 (0/124)

Data are presented as mean ± SD (range) and percent (number). RI indicates resistive index.

Table 2. Characteristics of SGA Fetuses Recognized Prenatally Versus Those Unrecognized Prenatally

Parameter	Unrecognized Prenatally	Recognized Prenatally	*P*
Maternal age, y	31.93 ± 5.45	31.51 ± 4.61	.461
Multiparity	37.0 (70/189)	25.8 (32/124)	<.05
GA, wk	39.3 ± 1.4	38.8 ± 1.7	<.05
Neonatal weight, g	2690 (2530–2850)	2575 (2315–2753)	<.05
Apgar score <7 at 1 min	0 (0/189)	1.6 (2/124)	.156
Apgar score <7 at 5 min	0.5 (1/189)	1.6 (2/124)	.565
pH	7.29 (7.22–7.32)	7.30 (7.24–7.33)	.095
Base excess, mEq/L	–3.88 ± 3.77	–3.62 ± 2.62	.692
Pco_2, mm Hg	52.81 ± 13.02	42.93 ± 26.33	.136
Po_2, mm Hg	20.91 ± 17.34	17.09 ± 6.73	.293
Fetal cord blood pH			
<7.00	0 (0/189)	0 (0/124)	>.99
<7.10	3.7 (7/189)	0 (0/124)	<.05
<7.15	10.1 (19/189)	1.6 (2/124)	<.05
Neonatal hospitalization, d	3.25 ± 1.68	3.08 ± 2.14	.459
NICU admission	4.8 (9/189)	3.2 (4/124)	.505
Medical induction of labor	30.2 (57/189)	41.9 (52/124)	<.05
Abnormal CTG	30.2 (57/189)	30.6 (38/124)	.927
Stained amniotic fluid	6.3 (12/189)	5.6 (7/124)	.799
Mode of delivery			
Spontaneous vaginal	58.7 (111/189)	46.8 (58/124)	<.05
Surgical vaginal	10.6 (20/189)	9.7 (12/124)	.796
Cesarean	30.7 (58/189)	43.5 (54/124)	<.05
GA at delivery			
34–36 wk	3.2 (6/189)	12.9 (16/124)	<.05
37–38 wk	24.9 (47/189)	25.8 (32/124)	.852
39–42 wk	72.0 (136/189)	61.3 (76/124)	<.05
Neonatal weight percentile			
<3	21.2 (40/189)	36.3 (45/124)	<.05
3–10	78.8 (149/189)	63.7 (79/124)	<.05

Data are presented as mean ± SD, median (interquartile range), and percent (number). CTG indicates cardiotocography.

Table 3. Reasons for NICU Admission and Neonatal Complications During NICU Hospitalization

Parameter	Unrecognized Prenatally	Recognized Prenatally	*P*
Reason for NICU admission			
Clinical suspicion of respiratory distress	33.3 (3/9)	0 (0/4)	.497
Jaundice[a]	44.4 (4/9)	50.0 (2/4)	>.99
Anemia[a]	22.2 (2/9)	0 (0/4)	>.99
Hypoglycemia	11.1 (1/9)	50.0 (2/4)	.203
Neonatal complications in NICU			
Intraventricular hemorrhage	0 (0/9)	0 (0/4)	>.99
Periventricular leukomalacia	0 (0/9)	0 (0/4)	>.99
Respiratory distress syndrome	22.2 (2/9)[b]	25.0 (1/4)	>.99
Necrotizing enterocolitis	0 (0/9)	0 (0/4)	>.99
Retinopathy of prematurity	0 (0/9)	25.0 (1/4)	.308
Neonatal sepsis	11.1 (1/9)	50.0 (2/4)	.203

Data are presented as percent (number).
[a]One neonate was affected by anemia and jaundice.
[b]One case of respiratory distress syndrome was caused by meconium aspiration.

fetal acidemia (pH <7.15) in unrecognized SGA fetuses in the 39- to 42-week group. Among fetuses hospitalized in the NICU, the prevalence of SGA at or below the 3rd percentile was 46.2% (6 of 13), and the prevalence of cesarean delivery was 23.1% (3 of 13).

Unrecognized SGA fetuses had worse outcomes than controls. Arterial cord pH values were significantly lower in unrecognized SGA fetuses than AGA fetuses regardless of medical induction of labor ($P < .05$), whereas arterial cord pH values were similar in recognized SGA and AGA fetuses, and medical induction of labor in unrecognized SGA fetuses significantly decreased the arterial cord pH values ($P < .05$). However, medical induction of labor in recognized SGA fetuses did not alter fetal outcomes significantly. Moreover, the prevalence of fetal acidemia (cord pH <7.15) was significantly lower in recognized SGA fetuses than AGA fetuses with medical induction of labor, whereas in unrecognized SGA fetuses, the prevalence of fetal acidemia was significantly higher than in AGA fetuses.

Table 5 shows reasons for medical induction of labor and cesarean delivery. Among the recognized SGA fetuses, those at or below the 3rd percentile had a higher prevalence of cesarean delivery ($P < .05$). The same pattern was observed in unrecognized SGA fetuses but did not reach statistical significance. We also found no significant differences among the studied groups for severe fetal acidemia (pH <7.00), 5-minute Apgar score less than 7, neonatal death before discharge, subdural hemorrhage, neonatal cerebral palsy, and stillbirth.

Discussion

This study indicates that prenatally recognized SGA, which requires strict antepartum surveillance and more careful management during labor, is related to a lower risk of adverse fetal outcomes compared to SGA fetuses who are recognized only after delivery. In our study, only 39.6% of SGA fetuses were prenatally recognized, and the actual detection rate at term was less than 50%. These findings could be explained by lack of fetal weight estimation or overestimation at the third-trimester growth scan.[20] Although umbilical artery Doppler sonography is effective in reducing perinatal death of preterm IUGR fetuses, it loses its ability near term, when the presence of diastolic

Table 4. Gestational Ages at Delivery and Fetal Outcomes

Parameter	Unrecognized Prenatally	Recognized Prenatally	*P*
34–36 wk			
Fetal cord blood pH			
<7.00	0 (0/6)	0 (0/16)	>.99
<7.10	16.7 (1/6)	0 (0/16)	.273
<7.15	16.7 (1/6)	0 (0/16)	.273
Neonatal hospitalization, d	6.75 ± 2.36	5.15 ± 3.74	.339
NICU admission	33.3 (2/6)	0 (0/16)	.065
Abnormal CTG	50.0 (3/6)	43.8 (7/16)	.793
Stained amniotic fluid	0 (0/6)	0 (0/16)	>.99
37–38 wk			
Fetal cord blood pH			
<7.00	0 (0/47)	0 (0/32)	>.99
<7.10	4.3 (2/47)	0 (0/32)	.512
<7.15	6.4 (3/47)	0 (0/32)	.268
Neonatal hospitalization, d	3.87 ± 1.45	3.42 ± 1.82	.255
NICU admission	2.1 (1/47)	3.1 (1/32)	.782
Abnormal CTG	27.7 (13/47)	43.8 (14/32)	.139
Stained amniotic fluid	0 (0/47)	6.2 (2/32)	.083
39–42 wk			
Fetal cord blood pH			
<7.00	0 (0/136)	0 (0/76)	>.99
<7.10	2.9 (4/136)	0 (0/76)	.299
<7.15	11.0 (15/136)	2.6 (2/76)	<.05
Neonatal hospitalization, d	2.93 ± 1.57	2.56 ± 1.59	.106
NICU admission	4.4 (6/136)	3.9 (3/76)	.872
Abnormal CTG	30.1 (41/136)	22.4 (17/76)	.223
Stained amniotic fluid	8.8 (12/136)	6.6 (5/76)	.564

Data are presented as mean ± SD and percent (number). CTG indicates cardiotocography.

flow might be related to a placental compensation mechanism in response to ischemia.[7] Recent evidence has demonstrated that a substantial proportion of SGA fetuses with normal umbilical artery Doppler values have milder forms of late-onset IUGR, as shown by an increased risk of adverse perinatal outcomes and abnormal neurodevelopment in the neonatal period and childhood.[4,5] Gestational age at delivery, abdominal circumference, oligohydramnios, and the cerebroplacental ratio in IUGR fetuses beyond 34 weeks' gestation are predictors of adverse fetal outcomes,[11,21] whereas the role of the ductus venosus in late-onset IUGR with normal umbilical artery Doppler values is still debated.[22] Finally, a short-term variation less than 3.5 milliseconds on computerized cardiotocography is most predictive of an umbilical artery cord pH lower than 7.20 at birth.[23] In this study, recognized SGA fetuses were defined according to Italian growth curves, had normal fetal and maternal Doppler values (umbilical artery, cerebroplacental ratio, ductus venosus, and uterine arteries) and regular computerized cardiotocographic analysis. Unrecognized SGA fetuses had normal umbilical artery Doppler values, but the status of the other arterial and venous vessels (cerebral artery and ductus venosus) was not known. This factor could explain the higher prevalence of an abnormal fetal heart rate in labor, pH lower than 7.15 or 7.10, longer hospitalization, and NICU admission in unrecognized SGA fetuses.

This study has shown that in prenatally recognized SGA fetuses, a policy of labor induction did not affect either the rate of adverse neonatal outcomes or the rate of instrumental vaginal or cesarean delivery. However, the rate of cesarean or surgical delivery was higher in the recognized and unrecognized SGA groups than in the AGA controls ($P < .05$). Moreover, prenatally recognized SGA fetuses with medical induction of labor were born more frequently by cesarean delivery than controls with medical induction of labor ($P < .05$). Induction of labor in recognized SGA fetuses was performed mainly for suspected growth arrest, reduced amniotic fluid, or an abnormal fetal heart rate tracing. In unrecognized SGA and AGA fetuses, the maternal status remains a more frequent indication for induction of labor than in recognized SGA fetuses, as previously described.[24] We confirm that SGA fetuses had a higher risk of cesarean delivery during labor than AGA fetuses even when the fetal and maternal Doppler values in our study were normal.[25] This result partly confirms previous observational studies of the roles of active management and antenatal surveillance in increased fetal monitoring and medical interventions.[24] In contrast to other studies[3,26] and in accordance with some authors, this

Table 5. Reasons for Medical Induction of Labor and Cesarean Delivery

Parameter	Unrecognized Prenatally Without Medical Induction	Recognized Prenatally Without Medical Induction	Unrecognized Prenatally With Medical Induction	Recognized Prenatally With Medical Induction	Control With Medical Induction	Control Without Medical Induction	*P*
Reason for induction							
Preterm PROM/PROM			42.1 (24/57)	19.2 (10/52)	36.6 (34/93)		<.05
Prolonged pregnancy (>41.4 wk)			8.8 (5/57)	1.9 (1/52)	17.2 (16/93)		<.05
Oligohydramnios			10.5 (6/57)	7.7 (4/52)	26.9 (25/93)		<.05
PRHDs			10.5 (6/57)	13.5 (7/52)	4.3 (4/93)		.141
Fetal growth arrest			0 (0/57)	42.3 (22/52)	0 (0/93)		<.05
Reduced fetal movements/ nonreassuring CTG			21.1 (12/57)	13.5 (7/52)	6.5 (6/93)		<.05
Other[a]			7.0 (4/57)	1.9 (1/52)	8.6 (8/93)		.284
Reason for cesarean[b]							
Fetal heart rate abnormalities	31.8 (14/44)	27.8 (10/36)	64.3 (9/14)	77.8 (14/18)	37.5 (6/16)	37.5 (6/16)	<.05
Labor dystocia	11.4 (5/44)	11.1 (4/36)	42.9 (6/14)	16.7 (3/18)	50.0 (8/16)	31.2 (5/16)	<.05
Fetal growth arrest	0 (0/44)	80.6 (29/36)	0 (0/14)	44.4 (8/18)	0 (0/16)	0 (0/16)	<.05
Previous cesarean	27.3 (12/44)	16.7 (6/36)	0 (0/14)	0 (0/18)	0 (0/16)	18.8 (3/16)	<.05
Other[c]	29.5 (13/44)	0 (0/36)	0 (0/14)	22.2 (4/18)	12.5 (2/16)	18.8 (3/16)	<.05

Data are presented as percent (number). CTG indicates cardiotocography; PRHD, pregnancy-related hypertensive disorder; and PROM, premature rupture of membranes.

[a]Other reasons included cholestasis, maternal diabetes, and hyperpyrexia.

[b]In some cases, there was more than 1 reason for cesarean delivery.

[c]Other reasons included hereditary muscular atrophy, orthopedic indication, psychiatric indication, hyperpyrexia, previous myomectomy, failed medical induction of labor, cholestasis, PRHDs, and tokophobia.

study showed that a suspicion of SGA followed by active management of labor and delivery resulted in a better neonatal outcome at birth in comparison to unrecognized SGA fetuses.[10,20,24,27] In fact, considering neonates delivered after 39 weeks' gestation, we found a significantly higher prevalence of pH lower than 7.15 in unrecognized than recognized SGA fetuses ($P < .05$; Table 4), but this difference was not significant among early gestations. As previously suggested, in contrast to our results, active management could be detrimental for constitutionally small fetuses who follow their own growth trajectory during pregnancy, and a recent randomized trial did not find significant differences in neonatal morbidity between induction and expectant management of suspected term SGA fetuses.[13] In particular, that study supports the hypothesis that the higher rate of NICU admission after induction of labor was a regular care-driven effect of an earlier GA and a lower birth weight rather than a result of defined complications, and if induction to avoid stillbirth is considered, it is reasonable to delay it until 38 weeks and provide continuous monitoring. In our study, the mean GA at delivery was 38 weeks, and the rate of NICU admission was higher in the 34- to 36- and 39- to 42-week ranges than in the 38- to 39-week range. Moreover, the fetuses with suspected growth restriction in the abovementioned trial were mixed: some had abnormal and others had normal Doppler values.[13] In our study of SGA fetuses with normal Doppler values, we did not observe major negative neonatal outcomes but only a few cases of hypoglycemia, hematological problems, respiratory distress syndrome, and meconium aspiration, which were solved during NICU admission. We did not find perinatal mortality or stillbirth, probably because of the absence of severe fetal acidemia (pH <7.00) and the low incidence of stillbirth after 34 weeks' gestation (0.2%).[28]

The main strength of this retrospective study is that it provides better insight into daily practice and describes a well-defined cohort of near-term and term SGA fetuses with normal fetal and maternal Doppler values. Limitations of this study were its retrospective nature and the number of cases.

In summary, this study indicates that prenatally recognized SGA after 34 weeks' gestation leads to more active management of labor and delivery. Identification of SGA fetuses resulted in better neonatal outcomes compared to unrecognized SGA fetuses, even if the mode of delivery was not modified between the two groups. Fetal and maternal antepartum surveillance has the advantage of lower perinatal morbidity (pH <7.10), although it does not lower surgical and cesarean delivery rates.

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