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Balancing the risks of stillbirth and neonatal death in the early preterm small-for-gestational-age fetus

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Abstract

Objective—Timing of delivery for the early preterm small for gestational age (SGA) fetus remains unknown. Our aim was to estimate the risk of stillbirth in the early preterm SGA fetus compared to the risk of neonatal death.

Study Design—We performed a retrospective cohort study of singleton pregnancies undergoing second trimester anatomy ultrasound excluding fetal anomalies, aneuploidy and pregnancies with incomplete neonatal follow-up. SGA was defined as birthweight < 10th percentile by the Alexander standard. Life-table analysis was used to calculate the cumulative risks of stillbirth/10,000 ongoing SGA pregnancies and risk of neonatal death/10,000 SGA live births for 2 week GA strata in the early preterm period (24–33 and 6/7 weeks). We further examined the composite risk of expectant management and then compared the risk of expectant management with the risk of immediate delivery.

Results—Of 76,453 singleton pregnancies, 7,036 SGA pregnancies meeting inclusion criteria were ongoing at 24 weeks with 64 stillbirths, 226 live births and 18 neonatal deaths between 24–33 and 6/7 weeks. As the risk of stillbirth increases with advancing GA, the risk of neonatal death falls, until the 32–33 and 6/7 week GA stratum. The relative risk of expectant management compared with immediate delivery remains <1 for each gestational age strata.

Conclusion—Our findings suggest the balance between the competing risks of stillbirth and neonatal death for the early preterm SGA fetus occurs at 32–33 and 6/7 weeks. These data can be useful when delivery timing remains uncertain.

Keywords

preterm; fetal growth restriction; IUGR; stillbirth; SGA

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Introduction

The small for gestational age (SGA) fetus carries an increased risk for stillbirth.¹ When SGA is diagnosed in the early preterm period, the clinician is faced with a difficult scenario. The decision to abandon expectant management and proceed to delivery in order to avoid stillbirth must, be delicately balanced with the risks of prematurity and neonatal death.

The first randomized trial on delivery timing in the preterm fetus estimated to be growth restricted demonstrated that early delivery to avoid stillbirth was counterbalanced by neonatal death.² Further, long-term follow-up showed no difference in childhood neurologic outcomes. This suggests that progressive fetal exposure to hypoxemia and acidemia with expectant management may not be associated with irreversible neurologic impairment as previously thought.³ Additionally, in a prospective study of fetuses estimated to be SGA with evidence of placental dysfunction, gestational age at delivery was demonstrated to be the dominant risk factor for neonatal outcomes relative to other risk factors for neonatal morbidity.⁴ Although the complications of prematurity weigh heavily on the decision to deliver, it is only the surviving fetus that will go on to become a neonate and the surviving neonate that will go on to experience the sequela of prematurity, making the first step in clarifying the optimal timing of delivery for the preterm SGA fetus to understand the competing risks of death, stillbirth and neonatal death. Therefore, the objective of our investigation was to estimate the risk of stillbirth compared to the risk of neonatal death in the early preterm SGA fetus.

Methods

We conducted a retrospective cohort study of singleton pregnancies presenting to Washington University School of Medicine perinatal ultrasound units for routine anatomic survey from 1990-2009. We utilized the perinatal database at Washington University. Our medical center is an academic tertiary care center that serves as a major regional and national referral center. Our perinatal database is a large, well maintained system with dedicated research personnel for the collection of data and maintenance. Self-report questionnaires are utilized to collect maternal demographic information, medical and obstetric history. The questionnaires are administered at the initial ultrasound visit. Follow-up information is obtained by trained research personnel from the medical record. In the event that the patient delivers outside of the medical system, follow-up is obtained through telephone contact with the patient and/or referring physician. Further details of data collection and management have been published previously.⁵

Ultrasounds were performed by certified sonographers dedicated to performing obstetric and gynecologic exams. Final diagnosis was made by the attending maternal fetal medicine physician. The gestational age was assigned by ultrasound dates if greater than five days from last menstrual period (LMP) in the first trimester, or greater than 10 days from LMP in the second trimester. SGA was assigned by birthweight using the population-based chart published by Alexander *et al*⁶ and defined as birthweight < 10th percentile for the gestational age at delivery. We excluded pregnancies complicated by prenatally diagnosed

major fetal anomalies and aneuploidy, and those without neonatal follow-up or birthweight data (figure 1).

Given our aim was focused on comparing the risks of stillbirth with neonatal death in the early preterm period, we examined the risks of stillbirth and neonatal death from 24-33 and 6/7 weeks utilizing the method of life-table analysis described by Smith *et al.*⁷ First, we calculated the risk of stillbirth by week. Within our database, stillbirth is defined as intrauterine fetal death at or beyond 20 weeks gestation but deliveries prior to 24 weeks or at 34 weeks and beyond were excluded from the analyses in accordance with the aim of this study. We calculated the conditional probability of stillbirth/10,000 ongoing SGA pregnancies. To account for censoring, deliveries that may have occurred during the particular time period for which the probability was calculated, 1/2 the number of deliveries during that time period were subtracted from the denominator. Therefore the conditional probability (**P**) of stillbirth (SB) during time period n given ongoing SGA pregnancies at the beginning of that time period n is (OP_n) and the number of births B : $P(SB)_n = SB_n / (OP_n - 1/2B_n)$.

The clinical question of timing of delivery is not limited to the conditional probability of stillbirth because if expectant management is chosen then the fetus remains in-utero during this time period and is exposed to the risks of stillbirth in the weeks preceding delivery. Therefore, the risk of stillbirth for the fetus at 28 weeks that is currently in-utero at 26 weeks is the cumulative probability of stillbirth at 26, 27 and 28 weeks. The cumulative probabilities of stillbirth were calculated from the conditional probabilities as 1-(probability of survival) where the probability of survival is 1-(probability of death). Therefore, the cumulative probability (**CP**) of stillbirth (SB) during time n : $CP(SB)_n = 1 - [(1 - C_{n1})(1 - C_{n2}) \dots (C_{n_x})]$.

In order to compare the risk of stillbirth to the risk of neonatal death over time, the risk of neonatal death was calculated per live births. Neonatal death was defined as death by 30 days of life. Neonatal deaths were relatively rare, therefore gestational age strata were collapsed into two week intervals. The conditional probability **P** of neonatal death (D) during time period n with live births (L) was calculated as: $P(D)_n = D_n / L_n$.

To compare the risk of neonatal death to the risk of stillbirth over time, we then collapsed stillbirths into 2 week strata and calculated the conditional and cumulative probabilities of stillbirth for each stratum. To explore a lower threshold of SGA, a secondary analysis was performed to evaluate the risks of neonatal death and stillbirth with SGA defined by birthweight <5th percentile.

Finally, because the cumulative probability is a retrospective calculation used to project risk into the future, and the point in time when SGA was diagnosed is not known, we also took a prospective approach to the probability of death by estimating the relative risk of expectant management for 2 weeks compared to immediate delivery as previously described by Rosenstein *et al.*⁸ Using this approach, the composite risk of expectant management for a time period is the sum of the conditional probability of stillbirth during that time period and

the probability of neonatal death in the following time period. This method assumes delivery in the subsequent interval of time.

Descriptive statistics were used to calculate maternal characteristics of SGA pregnancies delivering between 24-33 and 6/7 weeks for stillbirths, neonatal deaths and neonates surviving beyond 30 days of life. The cumulative risk of stillbirth with 95% confidence interval (CI) and risk of neonatal death with 95% CI were calculated for 2 week gestational age strata as stated above and then risk ratios with 95% CI were determined. Given the 20 year study period, sensitivity analysis was performed to assess changes in clinical practice or technology over time. The risk of stillbirth among SGA pregnancies delivering beyond 24 weeks was assessed and compared using χ^2 for two time periods determined by days from initiation of enrollment of 50% of the study cohort. Statistical analysis was calculated with STATA 12 (StataCorp, College Station, TX).

Results

Of 76,453 singleton pregnancies, there were 7,036 ongoing SGA pregnancies at 24 weeks meeting inclusion criteria and 290 SGA births between 24-33 and 6/7 weeks. Details of the study population and breakdown of the outcomes of SGA births occurring between 24-33 and 6/7 weeks gestation is located in figure 1. Table 1 demonstrates relevant maternal demographic characteristics of the ongoing SGA pregnancies delivering between 24-33 and 6/7 weeks resulting in stillbirth, neonatal death, or neonatal survival beyond 30 days of life.

The number of ongoing SGA pregnancies, stillbirths, live births and neonatal deaths per week is demonstrated in table 2 along with the conditional and cumulative probabilities of stillbirth/10,000 ongoing SGA pregnancies. With increasing gestational age, the cumulative risk of stillbirth rises from 14/10,000 ongoing SGA pregnancies in the 24th week of gestation to 91/10,000 ongoing SGA pregnancies in the 33rd week of gestation (figure 2).

The cumulative risk of stillbirth/10,000 ongoing SGA pregnancies and risk of neonatal death/10,000 SGA live births by 2 week gestational age strata is shown in table 3. There is a steep fall in neonatal deaths with increasing gestational age from 4,444/10,000 SGA live births during the 24-25 and 6/7 weeks strata to 89/10,000 SGA live births during the 32-33 and 6/7 weeks strata (figure 3). The cumulative risk of stillbirth over gestation as demonstrated in figure 2 is displayed again in figure 3 on a larger scale which also shows the risk of neonatal death over time. The risk of neonatal death is greater than the risk of stillbirth for all gestational age strata up to 31 and 6/7 weeks at which point the ratio of the risks is 0.97(table 3).

The composite risk of expectant management for 2 weeks compared with immediate delivery was calculated and results located in Table 4. The risk of expectant management was protective for each gestational age strata as demonstrated by relative risk < 1 for each 2 week strata.

Sensitivity analysis demonstrated no significant difference between the risk of stillbirth or neonatal death among the first 50% and second 50% of the enrolled cohort of ongoing SGA pregnancies at 24 weeks (102/10,000 vs. 80/10,000 (p=0.6) and 735/10,000 vs. 889/10,000

($p=0.7$)). Secondary analysis exploring a lower threshold of SGA, where SGA was defined as birthweight $< 5\%$ (Alexander *et al.*)⁶ found that 58/64 (90%) stillbirths had a birthweight $< 5\%$. The relationship between neonatal death and stillbirth over time continued to demonstrate an increased risk of neonatal death for each gestational age strata and the ratio of the risks of neonatal death to stillbirth reached 1.5 at the 32-33 and 6/7 week strata (Table 5).

Comment

By evaluating the risk of stillbirth relative to the risk of neonatal death, our data demonstrate a slow rise in the cumulative risk of stillbirth and a precipitous fall in the risk of neonatal death from 24-33 and 6/7 weeks. Risk ratios >1 for neonatal death compared to stillbirth indicate the risk of neonatal death was greater than the risk of stillbirth if delivery occurred between 24-31 and 6/7 weeks. Further, expectant management demonstrated a protective effect with relative risk < 1 .

The Growth Restriction Intervention Trial (GRIT) was designed to answer the question of timing of delivery for the early onset growth restricted fetus when the decision to deliver was unclear.² Initial results of the trial published in 2003 demonstrated no difference in mortality between expectant management and delivery. Follow-up of the GRIT cohort showed a trend toward more disability at 2 years in the immediate delivery group, but overall, no significant difference in death or severe disability with adjustment for gestational age and Doppler category.⁹ The authors noted that a majority of disability at 2 years occurred in the group delivering prior to 30 weeks. Specifically, the risk of cerebral palsy in the immediate delivery group at 24-30 weeks was 10%, consistent with previous studies, but the delayed delivery group in this same gestational age stratum had no cases of cerebral palsy, which the authors suggest may indicate a protective effect of delayed delivery. One of the major concerns at the time GRIT was designed was the fetal neurologic effect of progressive exposure to hypoxemia and acidemia with expectant management of fetal growth restriction. However, long-term neurobehavioral outcomes of children from GRIT was published in 2011, and demonstrated no difference at school age (6-9 years) in motor, cognition, language and behavior.³ Our study is consistent with the findings of GRIT in support of expectant management of the early preterm SGA fetus.

Although GRIT remains heavily criticized for several elements of the study design and long enrollment period, the outcomes of GRIT are supported by the works of several investigators over the past decade. In 2007, Baschat *et al.* demonstrated that among fetuses with placental dysfunction, gestational age was most predictive of neonatal death and intact survival compared to a myriad of other variables including multiple Doppler parameters and birthweight.⁴ Ductus venosus absent or reversed flow was not predictive of outcome until after the gestational age parameters for overall survival and intact survival had been reached, supporting the role of expectant management in the very early preterm growth restricted fetus.

In further support of GRIT regarding the long-term neurobehavioral outcomes there has been a paradigm shift regarding the theory surrounding the development of fetal growth

restriction. Through the work of several authors for which full review would not be possible in this limited discussion, the long held theory that fetal growth restriction leads to changes in blood flow that then affect neurodevelopment is no longer the accepted school of thought. The abundance of evidence that has come to light over the past decade through the work of investigators at the patient bedside and in the laboratory has led to the understanding that prior to clinically evident changes in fetal growth, there is a series of pre-clinical events resulting from placental insufficiency that lead to a re-distribution of fetal blood flow and changes in endocrine function and nutrient utilization.¹⁰ Once the pre-clinical events become severe enough that the fetus has undergone significant nutritional deficiency; abnormalities in fetal growth become detectable. This is exemplified by the most extreme event, when fetal growth restriction occurs as late as term and progression has not led to changes in umbilical artery flow abnormalities, abnormal changes in the brain are still present.¹¹ With evidence now strong that neurologic changes occur prior to clinically detectable fetal growth restriction, concerns for fetal neurologic deterioration secondary to worsening placental insufficiency and thought that intervention in the form of delivery can modulate this neurologic impairment are no longer valid. Therefore, expectant management in the fetus with reassuring biophysical status is further supported.

One of the major strengths of this study is the use of our large, validated database which allows us to evaluate the risks of rare outcomes such as fetal and neonatal death.¹² Our database is well maintained by trained personnel, and therefore, does not carry most of the limitations of an administrative dataset. Of further strength was the use of contemporary methods to estimate probabilities of death. To estimate the conditional probability of stillbirth, we utilized ongoing at risk pregnancies in the denominator and accounted for censoring. Our estimation of the cumulative probability of stillbirth was also important because it is clinically applicable.^{12, 13} Finally, because the cumulative probability of stillbirth is a retrospective calculation to prospectively estimate risk, we recognize potential criticism as it assumes the risk factor, SGA, was present from the earliest point in the risk calculation. First, from a theoretical perspective, this critique assumes that the increased risk of stillbirth in the SGA fetus is only present once fetal growth restriction becomes clinically detectable. It would then follow that the fetus that is destined to become pathologically small, but does not yet demonstrate clinically detectable fetal growth restriction, is not at increased risk for stillbirth. However, it is now clear that clinically detectable fetal growth restriction is the final common pathway for placental dysfunction that may carry its own increase in stillbirth risk and therefore this assumption is likely invalid. Additionally, to address this concern from an analytic perspective, we also took an approach that assumes the diagnosis of SGA only at the time of delivery and prospectively estimated the relative risk of expectant management compared to immediate delivery. Through this estimation, we demonstrate the protective effect of expectant management, further supporting the role of expectant management seen in our initial estimates of death.

There are limitations of our study that must also be considered. Although our database is large and well maintained, it is also limited in the information available. First, we recognize the importance of placental pathology to any study of stillbirth. Unfortunately, we do not have documentation of placental specimens, and the decision to pursue placental pathology is confounded by indication of the delivering attending physician. Second, Doppler

velocimetry and fetal biophysical assessment are an important component of the evaluation and management of suspected fetal growth restriction that we are unable to report on. Although our database spanning over 19 years did not demonstrate changes in technology or clinical management that have translated into changes in the stillbirth and neonatal death rates, significant changes in the application and interpretation of fetal Doppler assessment over this time period would make any meaningful conclusions from Doppler assessment and perinatal death rates difficult. Furthermore, the definition of fetal growth restriction continues to be widely debated, with the incorporation of a variety of vessels, combinations of vessels and cut-offs to define varying levels of pathology. The expected direction of bias could be in either direction and would depend on the criteria used to define abnormal Dopplers and the use of these parameters to determine timing of delivery. Additionally, given that our primary objective was to examine the competing risks of stillbirth and neonatal death in the early preterm period the investigation was designed up front to evaluate the risk of perinatal death from 24-33 and 6/7 weeks and we are unable to make conclusions beyond 33 and 6/7 weeks. Finally, in any study of stillbirth, the use of birthweight is limiting because the timing of stillbirth is usually difficult to ascertain and often remote from delivery. However, this discrepancy is partly mitigated by accounting for censoring in the conditional probabilities and of even less concern in this study because we evaluated stillbirth after 24 weeks, a point in gestation when most women would have the perception of fetal movement. Furthermore, the use of birthweight permits us to examine the direct association between SGA and death, whereas estimated fetal weight introduces issues of the accuracy of ultrasound to estimate fetal weight, and is an entirely different question.^{14, 15} Only after we have tested the direct association between SGA and death, as we have in this study, will improvement of our technical ability to predict SGA become relevant.

Our study suggests the balance between the risks of SGA stillbirth and neonatal death in the early preterm period occurs at 32-33 and 6/7 weeks. Prior to 32 weeks, the risk of neonatal death outweighs the risk of stillbirth. Keeping mindful that it is only the surviving neonate that will go on to develop the morbidities of prematurity, our findings suggest 32 weeks is the gestational age when the morbidity of prematurity becomes a relevant part of the equation balancing the risks of delivery with the risks of expectant management.

We recognize that fetal growth restriction is a heterogeneous clinical problem to which we have taken a large-scale epidemiologic approach at comparing the competing risks of death. Although we cannot offer practice recommendations as to the optimal gestational age for delivery of the preterm SGA fetus, or comment on individual case scenarios that would need to account for fetal biophysical status, our findings do offer a broad perspective into the risks of both expectant management and immediate delivery that would suggest expectant management of the SGA fetus to at least 32 weeks is the approach that carries the lowest risk of perinatal death.

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Figure 1. Flow diagram of study population

Flow diagram detailing the study population and the exclusion criteria to identify SGA pregnancies ongoing at 24 week gestation. The diagram also details the breakdown of birth outcomes occurring between 24-33 and 6/7 weeks.

Figure 2. Cumulative Risk of Stillbirth

Rise in the cumulative risk of stillbirth/10,000 ongoing SGA pregnancies from 24 weeks-33 and 6/7 weeks gestation.

Figure 3. Risk of stillbirth and neonatal death

The rise in the cumulative risk of stillbirth/10,000 ongoing SGA pregnancies from 24 weeks-33 and 6/7 weeks gestation transposed over the fall in neonatal death/10,000 SGA live births.

Table 1

Demographic characteristics of 24-33 and 6/7 week SGA (<10th percentile) stillbirths, neonatal deaths and survivors beyond 30 days of birth

Characteristic	Stillbirths (n=64)	Neonatal death (n=18)	Survivors (n=208)
Maternal age, y (SD)	29.5 (24.5,35)	30 (24,33)	29 (24,34)
Advanced maternal age, n (%)	17 (26.5)	1 (5.6)	45 (21.6)
Race, n (%)			
Black	26 (40.6)	8 (44.4)	101 (48.6)
White	26 (40.6)	7 (38.9)	86 (41.3)
Other	12(18.8)	3 (16.7)	21 (10.1)
Nulliparous, n (%)	23 (35.9)	8 (44.4)	111 (53.3)
Chronic hypertension, n (%)	4 (6.3)	3 (16.7)	26(12.5)
Preeclampsia, n (%)	8 (13.3)	8 (44.4)	110 (53.4)
Pregestational diabetes, n (%)	2 (3.1)	0 (0)	15 (7.2)
Gestational age at delivery *	26.6 (25.1, 30.4)	26.2 (25.6, 27.4)	32.0 (30.1, 33.1)

* Median (interquartile range)

Table 2SGA <10th percentile: SGA live births, neonatal deaths, stillbirths and cumulative risk of stillbirth per

Gestational age at delivery	Ongoing SGA pregnancies 24 weeks	SGA live births N= 226	Neonatal death N=18	SGA stillbirths N=64	Conditional probability SB/10,000 ongoing SGA pregnancies	Cumulative probability SB/10,000 ongoing SGA pregnancies
24-24 ^{6/7}	7,036	6	1	10	14	14
25-25 ^{6/7>}	7,020	12	7	14	20	34
26-26 ^{6/7}	6,994	8	2	10	14	48
27-27 ^{6/7}	6,976	16	4	4	6	54
28-28 ^{6/7}	6,956	9	0	2	3	57
29-29 ^{6/7}	6,945	11	2	3	4	61
30-30 ^{6/7}	6,931	21	0	9	13	74
31-31 ^{6/7}	6,901	31	1	5	7	81
32-32 ^{6/7}	6,865	49	1	4	6	87
33-33 ^{6/7}	6,812	63	0	3	4	91

Table 3

Risks of stillbirth, neonatal death and relative risk of neonatal death to stillbirth (SGA <10th percentile).

Gestational week	Cumulative risk of SB/ 10,000 ongoing SGA pregnancies (95% CI)	Risk of neonatal death/ 10,000 SGA livebirths (95% CI)	Ratio of risks of neonatal death to stillbirth
24-25 ^{6/7}	34 (24-48)	4,444 (4,346-4,542)	130.7
26-27 ^{6/7}	54 (41-70)	2,500 (2,415-2,586)	46.3
28-29 ^{6/7}	61 (47-78)	1,000 (942-1,060)	16.4
30-31 ^{6/7}	82(65-102)	192 (166-221)	2.3
32-33 ^{6/7}	92(74-113)	89(72-109)	0.97

Table 4SGA <10th percentile: Risks and relative risks of delivery and expectant management

Gestational week	Composite risk of expectant management $\times$ 2 weeks/10,000* (95% CI)	Risk of neonatal death/ 10,000 (95% CI)	RR of expectant management 2 weeks compared to delivery (95% CI)
24-25 ^{6/7}	2,534 (2,449-2,620)	4,444 (4,346-4,542)	0.63(0.61-0.66)
26-27 ^{6/7}	1,020 (961-1,081)	2,500 (2,415-2,586)	0.53 (0.50-0.56)
28-29 ^{6/7}	199 (173-228)	1,000 (942-1,060)	0.32 (0.28-0.36)
30-31 ^{6/7}	109 (90-131)	192 (166-221)	0.72 (0.62-0.84)

* Composite risk= risk of stillbirth at this gestational age + risk of infant death in the next strata

** Composite risk of expectant management relative to the risk of neonatal death in that strata

Table 5SGA <5th percentile: Risks of stillbirth, neonatal death and ratio of risks of neonatal death to stillbirth

Gestational week	Cumulative risk of SB/ 10,000 ongoing SGA pregnancies (95% CI)	Risk of neonatal death/ 10,000 SGA live births (95% CI)	Ratio of risks of neonatal death to stillbirth
24-25 ^{6/7}	48 (35-43)	3,333 (3241-3426)	69.8
26-27 ^{6/7}	78 (62-97)	3,846 (3750-3942)	49.3
28-29 ^{6/7}	86 (69-106)	1,250 (1186-1316)	14.5
30-31 ^{6/7}	115 (95-138)	454 (414-497)	4.0
32-33 ^{6/7}	126 (105-150)	192 (166-220)	1.5