

Previous SGA or preterm infant increases stillbirth risk

Surkan PJ, Stephansson O, Dickman PW, Cnattingius S. Previous preterm and small-for-gestational-age births and the subsequent risk of stillbirth. N Engl J Med. 2004;350:777-785.

OBJECTIVE

To investigate the association between small-for-gestational-age (SGA) or preterm birth and subsequent risk of stillbirth.

RESULTS

Odds ratios for subsequent stillbirth were 2.1 (95% confidence interval [CI], 1.6–2.8) in women with a first infant born SGA at term; 3.4 (95% CI, 2.1–5.6) in women with a first infant born SGA between 32 and 36 weeks; and 5.0 (95% CI, 2.5 to 9.8) in women with a first infant born SGA before 32 weeks.

EXPERT COMMENTARY

In this population-based, retrospective, observational study utilizing the Swedish Birth Registry, which included 410,021 women who delivered first and second consecutive singleton infants between 1983 and 1997, stillbirth rates ranged from 2.4 per 1,000 women who previously delivered an infant of “normal” size to 19.0 per 1,000 women who previously delivered a very preterm, SGA infant.

The association between fetal growth restriction and adverse perinatal outcome, including death, is well known. A recent hospital-based cohort study¹ showed that a significant proportion of unexplained antepartum fetal deaths can be related to growth restriction. A population-based study² found approximately 4 in 10 stillbirths have no known cause; fetal growth restriction comprised more than a third of this group.² This study shows that risk extends to the second pregnancy, implying a continuum of the underlying condition.

Abnormal uteroplacental and fetoplacental angiogenesis have been implicated in fetal growth restriction and preeclampsia. Randomized trials and meta-analyses pro-

vide strong evidence that antepartum fetal surveillance with umbilical arterial Doppler ultrasound in conjunction with fetal heart rate monitoring and biophysical profile can substantially reduce unexplained stillbirths in prenatally recognized growth-restricted fetuses. Surkan et al acknowledge this, and a recent review in this journal³ addressed this issue in terms of clinical practice.

Strengths and weaknesses of the study

The study population included nearly all the births in Sweden over a decade and a half; thus, sampling bias was not an issue.

The main limitation was that it was a retrospective study based on the Birth Registry and therefore could not provide all information relevant to fetal growth restriction and death in utero. Moreover, these databases are often incomplete.

A persistent challenge is our inability to reliably distinguish a baby who is growth-restricted from one who is merely small.

BOTTOM LINE

The birth of an SGA infant in the first pregnancy substantially increases the risk of antepartum fetal death in the subsequent pregnancy. Timely diagnosis of fetal growth restriction and appropriate evidence-based management may reduce this risk. ■

Risk of growth restriction extends to the second pregnancy, implying a continuum of the underlying condition.

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