

OBSTETRICS

Association between maternal characteristics, abnormal serum aneuploidy analytes, and placental abruption

Yair J. Blumenfeld, MD; Rebecca J. Baer, MPH; Maurice L. Druzin, MD; Yasser Y. El-Sayed, MD; Deirdre J. Lyell, MD; Alison M. Faucett, MD; Gary M. Shaw, PhD; Robert J. Currier, PhD; Laura L. Jelliffe-Pawlowski, PhD

OBJECTIVE: The objective of the study was to examine the association between placental abruption, maternal characteristics, and routine first- and second-trimester aneuploidy screening analytes.

STUDY DESIGN: The study consisted of an analysis of 1017 women with and 136,898 women without placental abruption who had first- and second-trimester prenatal screening results, linked birth certificate, and hospital discharge records for a live-born singleton. Maternal characteristics and first- and second-trimester aneuploidy screening analytes were analyzed using logistic binomial regression.

RESULTS: Placental abruption was more frequent among women of Asian race, age older than 34 years, women with chronic and pregnancy-associated hypertension, preeclampsia, preexisting diabetes, previous preterm birth, and interpregnancy interval less than 6 months. First-trimester pregnancy-associated plasma protein-A of the fifth percentile or less, second-trimester alpha

fetoprotein of the 95th percentile or greater, unconjugated estriol of the fifth percentile or less, and dimeric inhibin-A of the 95th percentile or greater were associated with placental abruption as well. When logistic models were stratified by the presence or absence of hypertensive disease, only maternal age older than 34 years (odds ratio [OR], 1.4; 95% confidence interval [CI], 1.0–2.0), pregnancy-associated plasma protein-A of the 95th percentile or less (OR, 1.9; 95% CI, 1.2–3.1), and alpha fetoprotein of the 95th percentile or greater (OR, 2.3; 95% CI, 1.4–3.8) remained statistically significantly associated for abruption.

CONCLUSION: In this large, population-based cohort study, abnormal maternal aneuploidy serum analyte levels were associated with placental abruption, regardless of the presence of hypertensive disease.

Key words: maternal characteristics, placental abruption, serum analytes

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Placental abruption, or bleeding into the decidua basalis, complicates approximately 0.5% of all pregnancies.¹⁻⁴ Placental abruption is largely a clinical diagnosis, suspected when gravidas present with vaginal bleeding or severe

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abdominal pain, often accompanied by uterine contractions or nonreassuring fetal heart rate patterns.⁵ Serious adverse neonatal outcomes have been linked with

placental abruption, most notably preterm birth, small-for-gestational-age weight, and even neonatal hypoxic ischemic encephalopathy and death in severe cases.^{6,7} Adverse maternal outcomes have also been associated with placental abruption including increased rates of operative delivery, a need for blood transfusion, and hysterectomy.⁴

Risk factors for placental abruption include chronic maternal medical conditions such as thyroid and hypertensive disease, fetal and placental etiologies such as multiple gestation and umbilical cord abnormalities, and obstetric factors such as preterm premature rupture of membranes (PPROM).^{8,9} Maternal risk factors for placental abruption may even predispose patients to the development of cardiovascular disease later in life.¹⁰

Noninvasive aneuploidy screening using maternal serum analyte and first-trimester nuchal translucency measurement has become a standard part of prenatal care for many pregnancies.^{11,12}

From the Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology (Drs Blumenfeld, Druzin, El-Sayed, and Lyell), and the Division of Neonatal and Developmental Medicine, Department of Pediatrics (Dr Shaw), Stanford University School of Medicine, Stanford; Genetic Disease Screening Program, California Department of Public Health, Richmond (Ms Baer and Drs Currier and Jelliffe-Pawlowski); and Division of Preventive Medicine and Public Health, Department of Epidemiology and Biostatistics, University of California, San Francisco, School of Medicine, San Francisco (Dr Jelliffe-Pawlowski), CA, and Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, University of Colorado School of Medicine, Aurora, CO (Dr Faucett).

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Reprints: Yair J. Blumenfeld, MD, Assistant Professor, Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, 193 Walker Hays Dr., Stanford University School of Medicine, Palo Alto, CA 94303. yairb@stanford.edu.

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All serum aneuploidy analytes (pregnancy-associated plasma protein A [PAPP-A]), total human chorionic gonadotropin (hCG), alpha fetoprotein (AFP), unconjugated estriol (uE3), and dimeric inhibin-A (INH) are directly or indirectly associated with placental function and pregnancy maintenance.¹³⁻¹⁷ Although abnormal analyte levels have been associated with placental disorders, including preeclampsia and intrauterine growth restriction (IUGR), their association with placental abruption remains inconclusive.¹⁸⁻²⁰

Our objective was to examine the association between placental abruption, maternal characteristics, and routine first- and second-trimester aneuploidy screening analytes among a large population-based cohort of women undergoing prenatal screening for fetal aneuploidy.

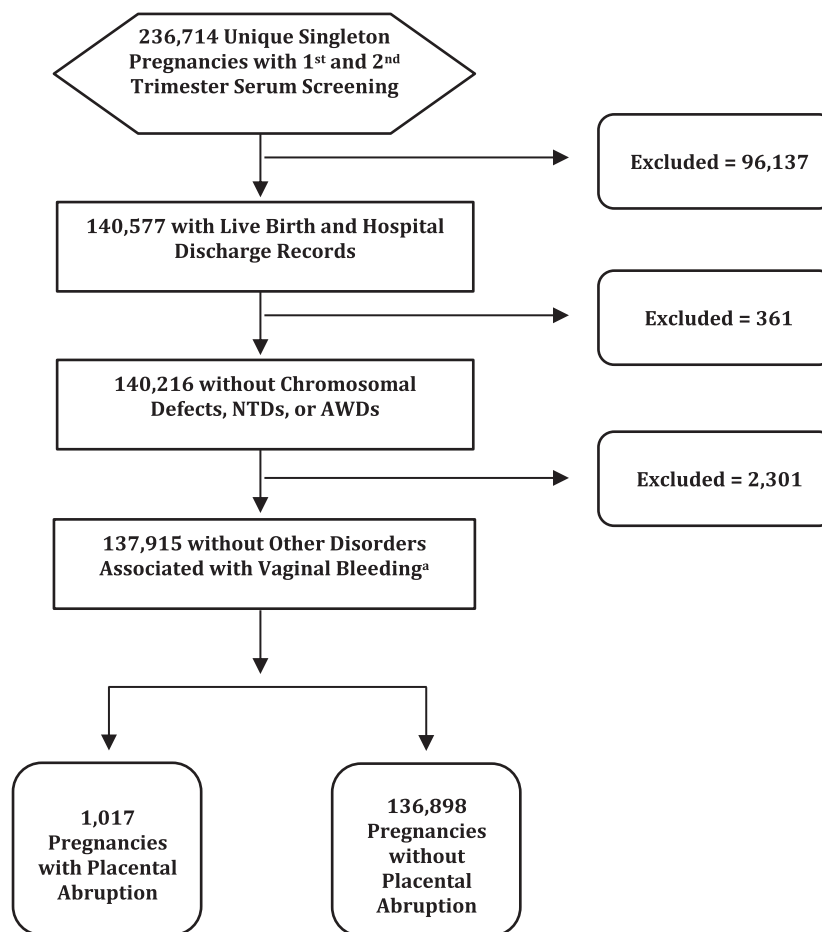
MATERIALS AND METHODS

The study sample was drawn from a cohort of 236,714 singleton pregnancies undergoing first- and second-trimester prenatal serum screening through the California Prenatal Screening Program administered by the Genetic Disease Screening Program (GDSP), with expected dates of delivery in 2009 and 2010. The sample was restricted to pregnancies that had a linked live birth and hospital discharge record in the birth cohort database maintained by the Office of Statewide Health Planning and Development (total with linked records = 140,577).

Pregnancies with fetal chromosomal abnormalities, neural tube defects, or abdominal wall defects were excluded ($n = 361$). To focus on placental abruption, pregnancies with other potential etiologies for vaginal bleeding were also excluded, including placenta previa and retained placenta without abruption ($n = 2301$). Of the 137,915 remaining pregnancies, 1017 experienced placental abruption and 136,898 did not (Figure 1).

Analyte results were derived from blood samples collected between a gestation of 10 weeks 0 days and 13 weeks 6 days in the first trimester, and a gestation of 15 weeks 0 days and 20 weeks 0 days in the second trimester. First-trimester

FIGURE 1
Overview of sample selection



^aPlacental previa and retained placenta without abruption.

Blumenfeld. Association between serum analytes and abruption. *Am J Obstet Gynecol* 2014.

analyte measurements included PAPP-A and hCG. Second-trimester analytes included AFP, hCG, uE3, and INH. Analyte levels were measured on automated equipment (Auto DELFIA; Perkin Elmer Life Sciences, Waltham, MA, and Applied Biosystems, Brea, CA), and results were entered directly into a state database along with patient information used to adjust multiple of the median (MoM) values associated with biomarker results and/or used in final result interpretation. All analyte MoMs were adjusted for gestational age, maternal weight (as a proxy for blood volume), race/ethnicity, smoking status, and preexisting diabetes. Data related to chromosomal, neural tube, and abdominal wall defects were obtained from the GDSP screening

records and associated defect registries. Details regarding the program and associated registries have been described elsewhere.^{11,21}

Maternal body mass index (BMI) was calculated using height (height²) and prepregnancy weight provided in the linked vital statistics birth and hospital discharge records. The interpregnancy interval was calculated from previous live birth (month and year) as reported in linked records and estimated as months to conception of the index pregnancy. Given that the day of the previous live birth was not available, the first of the month was used for calculation purposes. Parity, previous cesarean section, and previous preterm birth were also obtained from linked birth and hospital

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