

(CS), or transvaginally. In CS packing was generally combined with B-Lynch and/or Pereira stitches. 15036 consecutive births before and after the introduction of Celox (n=5498 vs. n=9538 deliveries) were analyzed. 1-tailed Fisher exact test was used for statistics.

RESULTS: Celox was used in 65 cases of PPH including 21 severe cases where ppHE seemed inevitable. 35 women had delivered vaginally (1 vacuum), 27 by elective (n=13) or emergency (n=14) CS. In 6 out of 35 vaginal deliveries laparotomy was necessary to apply compression sutures. Celox was left in utero for up to 48 hrs (mean 20.63) before extraction. Compared with 26 mth before, in the 28 mth after introduction of Celox the rate of ppHEs was significantly reduced (10 vs. 5; OR 3.47, 95% CI 1.19-10.16; p=.023). Indications for HEs were uterine rupture (n=2), failure of compression sutures (n=2, in one case no Celox was used), recurrence of PPH after removal of Celox after 48 hrs due to preexisting thrombopenia (18 thr per nl, n=1). The longest period without ppHE after Celox was 23.5 months. One of the Celox treated patients is pregnant again (34 wks gestation). Maternal mortality after the introduction of Celox was 0.

CONCLUSION: Celox is a viable option in the treatment of PPH and reduces ppHE significantly. It can safely be used after both vaginal and CS, and we observed no specific treatment associated morbidity. It is inexpensive compared to other treatments, making it suitable for use also in low resource-countries, where the death toll due to PPH is high.

735 Perinatal outcomes in pregnancies with a low fetal fraction on noninvasive prenatal testing results

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OBJECTIVE: We aimed to assess risk of adverse perinatal outcomes for pregnant women with a low fetal fraction compared with women who had a sufficient fetal fraction on noninvasive prenatal testing (NIPT) results.

STUDY DESIGN: We examined women undergoing NIPT between July 2012 and October 2013 at a tertiary care center. Inclusion criteria were singleton pregnancy and availability of pregnancy outcome information. We excluded women with aneuploidy, loss to follow-up, and those who underwent pregnancy termination.

RESULTS: 219 women who were eligible for inclusion underwent NIPT during the study period, 207 (94.5%) had a sufficient fetal fraction and 12 (5.5%) had a low fetal fraction result. The mean maternal age at delivery and gestational age at the time of NIPT were comparable for both groups. The leading indication for NIPT in both groups was advanced maternal age. The women who had a low fetal fraction result were more likely to be African-American (83%vs.31%;p=0.001) and had a significantly higher BMI (mean(SD) BMI=36(7.8) kg/m²vs. BMI=27(6.9) kg/m²;p<0.001) than the women who had a sufficient fetal fraction result on initial NIPT. 19.2% of African-American with a BMI≥35 had a low fetal fraction result. The risk of having a miscarriage, fetal demise, or neonatal death was significantly higher in the low fetal fraction group (16.7%vs.1.9%;p=0.037). However, after adjusting for race and/or BMI this association was no longer statistically significant. Rates of hypertensive disorders, low birthweight, preterm labor, placenta previa, and placental abruption were not different between groups.

CONCLUSION: Women with higher BMI's and of African-American descent were more likely to have a low fetal fraction result. Given the increased background risk of adverse perinatal outcome among obese African-American women, this association with a low fetal fraction warrants further investigation. Additional counseling regarding the possibility of a low fetal fraction result in this patient population should be considered.

Table 1 Maternal characteristics and pregnancy outcome among women with a low fetal fraction result

Characteristic/Outcome	Low Fetal Fraction (n=12)	Sufficient Fetal Fraction (n=207)	p value
Maternal age at delivery mean years (SD)	35.9 (5.4)	36.9 (4.1)	0.736
Gestational age at time of NIPT mean weeks (SD)	14.6 (3.7)	13.9 (3.6)	0.529
Race % (n)			
White	8.3 (1)	48.8 (101)	0.001
African-American	83.3 (10)	30.9 (64)	
Asian	-	15.9 (33)	
Other	8.3 (1)	2.4 (5)	
Missing	-	1.9 (4)	
BMI at time of NIPT mean kg/m ² (SD)	36.0 (7.8)	27.1 (6.9)	<0.001
Pregnancy outcome % (n) (miscarriage, fetal demise, or neonatal death)	16.7 (2)	1.9 (4)	0.037

736 Placental tumor necrosis factor- α levels throughout gestation in normal pregnancy

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OBJECTIVE: Placental Tumor Necrosis Factor- α (TNF α) is postulated to induce a cascade of events that confers invasive characteristics to the cytotrophoblasts and actively participates in apoptosis of vascular smooth muscle cells that surround the spiral arteries of maternal decidua, resulting in remodeling of the spiral arteries. We investigated placental TNF α levels throughout gestation in women with uncomplicated pregnancies.

STUDY DESIGN: In an IRB approved study, placental tissues were obtained from normal pregnant women who underwent elective abortion in first and second trimester of pregnancy, and from women who delivered at term. Specimens were collected within 10 minutes of completion of the procedure and dissected in saline to identify chorionic villi without associated decidua. Villous samples were stored at -80°C until assay. TNF α levels were determined using kits from Cayman Chemicals (Item # 589201, Ann Arbor, MI) and results were expressed as pg/100 mg tissue. Gestational age (GA) was assessed by ultrasound.

RESULTS: 160 placentas were grouped as: 1st trimester (≤ 13 weeks of GA), 2nd trimester (13⁺ to 26 weeks of GA), and 3rd trimester (26⁺ weeks of GA). The mean \pm SD of the TNF α levels are shown in Table 1. Kruskal Wallis test revealed significant differences in TNF α levels between the groups (H=13.301, p<0.001). Mann Whitney U test results showed that 2nd trimester TNF α levels were significantly different from that of the 1st (p<0.01) and 3rd trimester values (p<0.001). Spearman's Bivariate correlation showed a negative correlation between TNF α and gestational age in days (r=-0.092, p=0.249).

CONCLUSION: Placental TNF α levels peaked during the 2nd Trimester of normal pregnancy. It may be suggested that such a TNF α peak may play a role in the second wave of trophoblastic invasion and in remodeling of the spiral arteries. A failure in the induction of such a 2nd trimester increase in TNF α levels during pregnancy could compromise a successful pregnancy outcome and may also contribute to the development of preeclampsia.

Table 1 Placental TNF α expression throughout normal pregnancy

Groups	N	TNF α
		(pg / 100 mg tissue) Mean \pm SD
1 st Trimester	85	37.87 \pm 29.63
2 nd Trimester	46	56.53 \pm 40.81
3 rd Trimester	29	26.83 \pm 29.25