FELLOWS

Abstracts 42-49

Moderators: Mary D'Alton, MD; Yoel Sadovsky, MD

42 Torsemide for prevention of persistent postpartum hypertension in preeclampsia: a randomized, placebo-controlled trial

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OBJECTIVE: Postpartum hypertension (pHTN) in preeclampsia (PE) results, in part, from mobilization of extravascular fluid accumulated in pregnancy. Our objective was to determine whether the loop diuretic torsemide reduces the rate of persistent pHTN in women with PE.

STUDY DESIGN: Pragmatic, randomized, double-blind, placebo-controlled trial (RCT, NCT02813551) of women with PE or superimposed PE (with or without severe features) at a single center from August 2016 to July 2017. Those with chronic or gestational HTN, renal or cardiopulmonary failure were excluded. Within 24h of delivery, women were randomized to oral torsemide 20 mg/d or placebo, for 5d. Primary outcome was rate of persistent pHTN (sustained SBP≥150 or DBP≥100 mmHg) by postpartum day 5 or at hospital discharge, whichever occurred first. Secondary outcomes included: pHTN 7-10d after delivery, severe HTN (SBP≥160 or DBP≥110 mmHg), length of puerperal hospital stay, readmission for HTN, adverse events and severe composite morbidity (ICU admission, HELLP syndrome, eclampsia, stroke, renal/heart failure, pulmonary edema or death). A 50% rate of pHTN in the placebo arm was expected based on prior studies. Assuming a 25% rate with torsemide, 80% power and alpha-error of 0.05, a sample of 118 women was required. Bayesian analysis to calculate probability of treatment benefit or harm was planned *a priori*. Analyses were based on intention-to-treat.

RESULTS: 59 women were allocated to torsemide and 59 to placebo. Baseline characteristics were similar. Overall, 43 (73%) women in the torsemide group and 45 (76%) in the placebo group had either PE with severe features or superimposed to chronic HTN. The rate of persistent pHTN was 44% in torsemide vs 57% in placebo group (RR 0.76, 95% CI 0.53-1.10). However, Bayesian analysis revealed that probability of torsemide reducing persistent pHTN compared to placebo was 92%. There were no differences in rate of pHTN 7-10d after delivery, severe HTN, length of stay, readmission for HTN or adverse events (Table). There were no cases of severe composite morbidity or deaths.

CONCLUSION: In this RCT of women with PE, frequency of persistent pHTN was not reduced by torsemide. However, since Bayesian analysis reveals a likelihood of 92% that torsemide is superior to placebo in preventing the primary outcome, larger trials with longer duration of treatment are warranted.

Table: Study outcomes according to treatment group

	Torsemide (N = 59)	Placebo (N = 59)	RR (95% CI)	P value
Primary outcome*	26 (44.1)	34 (57.6)	0.76 (0.53-1.10)	0.14
HTN at 7-10 days postpartum	9 (15.3)	11 (18.6)	0.82 (0.37-1.81)	0.62
Postpartum Severe HTN†	13 (22.0)	18 (30.5)	0.72 (0.39-1.33)	0.30
Length of stay after delivery (hours)	67.3 (52.2-94.5)	54.4 (46.9-76.6)	-	0.09
ER visit for HTN	4 (6.8)	2 (3.4)	-	0.68
Readmission for HTN	3 (5.1)	1 (1.7)	-	0.62
Readmission < 7 days	0 (0.0)	1 (1.7)		1.00
Adverse Events				
Hypokalemia (< 3.5 mEq/dL)	1/22 (4.6)	4/31 (12.9)	-	0.39
Reduced breastmilk (7-10 days postpartum)	1 (1.7)	0 (0.0)	-	1.00

Data expressed as n (%), mean (interquartile range) as appropriate.
* Primary Outcome: Persistent HTN (sustained SBP ≥ 150 mmHg and/or DBP ≥ 100, or both) by postpartum day 5 or hospital discharge, whichever occurred first.
† Postpartum Severe HTN: SBP ≥ 160 mmHg and/or DBP ≥ 110 mmHg.

43 Contraction associated maternal heart rate decelerations: a pragmatic marker of intrapartum volume status

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OBJECTIVE: Echocardiography has demonstrated uterine contractions increase cardiac preload and stroke volume through auto-transfusion. These hemodynamic changes may be associated with a compensatory decrease in maternal heart rate. Our objective was to evaluate a visual maternal Contraction-Associated Heart Rate Deceleration (CAHD) pattern as a potential marker of relative hypovolemia.

STUDY DESIGN: We performed a pre-specified secondary analysis of a prospectively collected cohort from a RCT of maternal admission pulse pressure (PP), epidural coload volume, and post-epidural complications. Participants were healthy laboring women with non-anomalous singleton gestations ≥35 weeks and a category 1 FHR pattern from admission to epidural placement. Those without continuous maternal pulse oximetry data were excluded. Maternal heart rate tracings were evaluated for the presence of CAHD (Figure 1) in the hour prior to epidural placement. Women with and without CAHD were compared. Evaluated study outcomes were: post-epidural category 2/3 FHR abnormalities, maternal hypotension, and obstetric interventions.

RESULTS: Of 414 participants, 388 (93.7%) met inclusion criteria; of these 124 (32.0%) had CAHD before epidural placement. On admission, women with CAHD had lower systolic (116.6 vs 119.5, p=.002) and higher diastolic (75.3 vs 73.6, p=.03) blood pressures and a narrower PP (41.3 vs 46.0, p<.001) than women without CAHD. Demographic and clinical characteristics were otherwise similar between groups. CAHD was associated with more frequent post-epidural FHR abnormalities, diastolic hypotension, and obstetric interventions (Table 1). Among women with CAHD, a larger initial epidural bolus (1500 vs 500 mL) was associated with less frequent systolic hypotension (9.8 vs 32.8%, p=.003) and obstetric interventions (21.6 vs 42.5%, p=.02). Among women with CAHD and narrow PP, post-epidural FHR abnormalities were less frequent with a 1500 mL bolus (37.3 vs