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266 Metabolomic biomarkers of chronic hypertension in pregnancy

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compared. The primary outcome was need for neonatal resuscitation at delivery. Secondary outcomes included administration of beta-methasone, FGR and neonatal outcomes.

**RESULTS:** Of 527 women meeting study criteria, 42 had oligohydramnios that resolved prior to delivery, while 485 had persistent oligohydramnios. There were no significant differences in patient demographics or medical comorbidities between groups. The gestational age at diagnosis was significantly lower for patients with resolved versus persistent oligohydramnios (median 33 (interquartile range (IQR) 29.1-35.9) vs. 38.0 (IQR 36.4-39.3);  $p < 0.001$ ). There was no difference in neonatal resuscitation (41% vs. 32%,  $p=0.31$ ). Patients with resolved oligohydramnios were more likely to develop FGR than those with persistent oligohydramnios (55% vs. 36%  $p < 0.02$ ). There were no significant differences for rates of betamethasone administration, gestational age at delivery, birth weight, or NICU admission.

**CONCLUSION:** Patients whose oligohydramnios resolved were diagnosed at early gestational ages and had similar rates of neonatal resuscitation but higher rates of FGR than those who had persistent oligohydramnios. These findings suggest that women with oligohydramnios diagnosed early in the third trimester should be monitored closely for FGR.

Outcomes of Persistent vs Resolved Oligohydramnios			
	Persistent Oligohydramnios (N=485)	Resolved Oligohydramnios (N=42)	P-value
<b>Primary Outcome</b>			
Composite Neonatal Resuscitation* N (%)	157 (32.4)	17 (40.5)	0.31 <sup>2</sup>
<b>Secondary Neonatal Outcomes</b>			
Gestational Age at diagnosis of Oligo			<0.001 <sup>1</sup>
Mean (SD)	37.3 (3.4)	31.2 (6.3)	
Median (Min-Max)	38.0 (16.0-45.7)	33.0 (16.6-38.9)	
IQR (Q1-Q3)	(36.4-39.3)	(29.1-35.9)	
Betamethasone Administration N (%)	50 (10.4)	6 (14.3)	0.43 <sup>2</sup>
Fetal Growth Restriction N (%)	176 (36.3)	23 (54.8)	0.02 <sup>2</sup>
Gestational age at delivery			0.45 <sup>1</sup>
Mean (SD)	38.1 (2.0)	38.4 (1.7)	
Median (Min-Max)	38.3 (26.6-41.9)	39.0 (34.1-40.9)	
IQR (Q1-Q3)	(37.1-39.4)	(37.3-39.6)	
Birthweight (grams)			0.86 <sup>1</sup>
Mean (SD)	2848 (671)	2846 (613)	
Median (Min-Max)	2870 (410-4700)	2860 (1740-4455)	
IQR (Q1-Q3)	(2430-3300)	(2445-3360)	
5-minute Apgar <5 N (%)	3 (0.57)	0	1.00
NICU Admission N (%)	110 (22.7)	7 (16.7)	0.44 <sup>2</sup>
Diagnosis of RDS N (%)	33 (6.8%)	1 (2.4%)	0.50 <sup>2</sup>
Neonatal death N (%)	3 (0.62)	0	1.00 <sup>2</sup>
<b>Secondary Obstetric Outcomes</b>			
Type of Labor N (%)			<0.001 <sup>2</sup>
Spontaneous	10 (2.1)	7 (16.7)	
Spontaneous/augmented	21 (4.3)	4 (9.5)	
Pre-labor CS	123 (25.4)	9 (21.4)	
Induction	331 (68.3)	22 (52.4)	
Mode of Delivery N (%)	(n=479)	(n=42)	0.14 <sup>2</sup>
Vaginal	229 (47.8)	25 (59.5)	
Cesarean	229 (47.8)	14 (33.3)	
OVD	21 (4.4)	3 (7.1)	

<sup>1</sup>Wilcoxon rank-sum

<sup>2</sup>Fisher's exact test

\*Includes oxygen, bag and mask with oxygen, CPAP, intubation, chest compressions and cardiac medications

## 265 Hemivertebra: systematic review of cases with cytogenetic abnormalities, associated anomalies and proposed prenatal management

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**OBJECTIVE:** To identify cases of hemivertebra with abnormal cytogenetic studies and assess for other associated anomalies in order to provide more accurate patient counseling and prenatal management.

**STUDY DESIGN:** This systematic review was conducted using PubMed (including Cochrane database) and Ovid Medline from inception through November 2019. Studies were deemed eligible for inclusion if hemivertebra with abnormal cytogenetic studies was identified in the fetal, neonatal, or infant period. Studies that described other vertebral anomalies were excluded.

**RESULTS:** We identified 17 cases of hemivertebra with cytogenetic abnormalities among a total of 324 cases (5%) in our review. These include partial tetrasomy 4q, mosaic trisomy 4, mosaic trisomy 7, mosaic trisomy 9, mosaic trisomy 18q, mosaic trisomy 18; trisomy 7, trisomy 15q with monosomy 6q, partial trisomy 22; duplication of 2p; 4p- deletion, 17p deletion, 18p deletion, 18q22.2 deletion, 22q13.3 deletion; ring chromosome 21, and Fanconi's anemia. 29% (5/17) were diagnosed prenatally, 47% (8/17) at birth, and 18% (3/17) postnatally. All cases had prenatally or postnatally diagnosed associated anomalies. 29% (5/17) had associated anomalies identified prenatally, including ear anomalies, CNS abnormalities, micrognathia, congenital heart defects, renal anomalies, clubfeet, and fetal growth restriction. Postnatally diagnosed anomalies commonly included: CNS (41%, 7/17), cardiac (53%, 9/17), skeletal (71%, 12/17) and genitourinary (59%, 10/17).

**CONCLUSION:** Hemivertebra with abnormal cytogenetic testing was always associated with malformations from other organ systems that could be missed by prenatal ultrasound alone. Those include CNS, cardiovascular, musculoskeletal, and genitourinary anomalies. Fetal echocardiogram is recommended due to the considerable association with congenital heart defects. Fetal neurosonography or MRI should be considered. Of those receiving genetic testing, karyotype or SNP microarray should be considered due to the common presence of mosaicism.

## 266 Metabolomic biomarkers of chronic hypertension in pregnancy

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**OBJECTIVE:** Chronic hypertension is associated with adverse risks for the mother and fetus in pregnancy. Metabolomic profiling can help elucidate disease pathways. Free fatty acid (FFA) levels are elevated in hypertensive non-pregnant adults, and FFAs appear to impair microvascular function and contribute to microangiopathy. The objective of this study is to compare metabolomic biomarkers of chronic hypertension in a pregnant cohort.

**STUDY DESIGN:** We prospectively collected fasting serum samples from pregnant patients at 24-28 weeks gestation from 5 patients who had well-controlled chronic hypertension not on anti-hypertensive medications and 28 controls. These samples were then processed and evaluated using Q-TOF mass spectrometry and analyzed using a targeted protocol of 25 types of free fatty acids known to be present in people with hypertension. We used Student's t-test to compare metabolites between the two study groups.



**RESULTS:** Maternal age, parity, BMI, gestational age at delivery, neonatal birthweight, and APGAR scores were similar between the two groups. None of the patients developed preeclampsia or required acute treatment for hypertension. Accounting for Bonferroni correction, we found statistically significant elevations of 1.45-2.03 fold in 9 of the targeted unsaturated fatty acids in the chronic hypertension group compared to controls.

**CONCLUSION:** Using metabolomic profiling, we found that 9 unsaturated fatty acid were elevated in the serum of fasting pregnant women with chronic hypertension. This free fatty acid profile mirrors that of people with chronic hypertension outside of pregnancy. This may represent a possible pathway explaining how pregnant women with well controlled chronic hypertension remain at high risk for complications such as preeclampsia. Larger studies are needed to compare the fatty acid profile in women with chronic hypertension who developed pre-eclampsia.

Free fatty acid chemical formula	Fold change (Hypertension/control)	P
C16:0 (area)	1.63	<0.01
C16:1	1.85	0.03
C18:1	1.56	<0.01
C18:2	1.71	<0.01
C18:3	1.79	0.05
C20:3	2.03	<0.01
C20:4	1.45	0.03
C22:5	1.78	<0.01
C22:5 isomer	1.89	<0.01

\* p-value with Bonferroni correction

**267 Multiple fetal anomalies are associated with actionable postnatal genome sequencing results**

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**OBJECTIVE:** Prenatal whole exome sequencing (WES) and whole genome sequencing (WGS) have not been adopted into routine obstetrical practice due to high cost and suspected low diagnostic yield. However, the actual diagnostic yield of these methods in pregnancies with specific prenatal findings is not known. The goal of this study is to determine whether specific types of prenatal complications are associated with actionable postnatal WES/WGS results.

**STUDY DESIGN:** This is a case control study comparing critically ill neonates in the NSIGHT2 study who had actionable versus negative postnatal WES/WGS results. We excluded neonates without available prenatal ultrasound reports. We reviewed the ultrasound reports for abnormal findings. We calculated the odds ratio and used Fisher's exact test to compare findings between the two groups.

**RESULTS:** 213 neonates were sequenced in the NSIGHT2 study. 80 of those neonates had available prenatal ultrasounds. Of these, 21 had an actionable finding from genome sequencing: 7 WES and 14 WGS. 59 neonates had negative sequencing results: 32 WES and 27 WGS. 66.7% of neonates with actionable WES/WGS results had anomalies

suspected on fetal ultrasound, while 55.9% of neonates with negative sequencing had suspected fetal anomalies. Among those with suspected fetal anomalies, neonates with actionable WES/WGS results were 4.5 times more likely to have multiple anomalies and 6.7 times more likely to have anomalies of the extremities compared to those with negative findings. Fetal growth restriction was present in 19% of neonates with actionable WES/WGS and in 6.8% with negative sequencing results.

**CONCLUSION:** In our study population, neonates with actionable findings on WES/WGS were statistically more likely to have multiple anomalies and anomalies of the extremities. These findings suggest that pregnancies with multiple fetal anomalies on ultrasound may benefit from WES/WGS. Larger studies are needed to determine utility of introducing WES/WGS to clinical practice.

	Positive Results <sup>a</sup>	Negative Results <sup>a</sup>	OR (95% CI)	P <sup>b</sup>
Central Nervous System	7 (33.3)	9 (15.3)	2.78 (0.88-8.79)	0.11
Face	2 (9.5)	0	N/A <sup>c</sup>	0.07
Heart	7 (33.3)	21 (35.6)	0.91 (0.32-2.59)	1.0
Chest	1 (4.8)	2 (3.4)	1.43 (0.12-16.6)	1.0
Gastrointestinal	2 (9.5)	3 (5.1)	1.97 (0.31-12.7)	0.60
Genitourinary	5 (23.8)	5 (8.5)	3.38 (0.87-13.1)	0.12
Extremities	4 (19)	2 (3.4)	6.71 (1.13-39.8)	0.04*
Spine	1 (4.8)	0	N/A <sup>c</sup>	0.26
Umbilical Cord	3 (14.3)	4 (6.8)	2.29 (0.47-11.2)	0.37
Placenta	1 (4.8)	0	N/A <sup>c</sup>	0.26
Amniotic Fluid Index	4 (19)	6 (10.2)	2.08 (0.52-8.25)	0.44
Other Body System	0	2 (3.4)	N/A <sup>c</sup>	1.0
Any Anomaly	14 (66.7)	33 (55.9)	1.58 (0.56-4.47)	0.45
Multiple Anomaly	10 (47.6)	10 (16.9)	4.46 (1.49-13.3)	0.01*
Fetal Growth Restriction	4 (19)	4 (6.8)	3.24 (0.73-14.3)	0.20

<sup>a</sup> Data presented as N (percentage)  
<sup>b</sup> Analysis by Fisher's exact test  
<sup>c</sup> Not estimable due to zero cells  
 \* Statistically significant

**268 Utility of the INCODE algorithm to identify cause of stillbirth in clinical settings**

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**OBJECTIVE:** The Initial Causes of Fetal Death Evaluation (INCODE) algorithm was developed by the Stillbirth Collaborative Research Network (SCRN) as a research tool to assign causes of death of stillbirth in which standardized postmortem examination (PME) is performed. Our goal was to assess the utility of INCODE in a retrospective cohort of stillbirths (SB) at a single tertiary hospital where PME was not routinely performed.

**STUDY DESIGN:** Chart reviews were performed for all cases of SB from Dec 2013 to Oct 2019. Narrative summaries and PME results were recorded. Cause of death was assigned based on INCODE as described by SCRN investigators.

**RESULTS:** Of the 99 identified SB, probable cause of death was identified in 70 (70.7%; 95% CI, 61.7-79.7%) and possible or probable cause in 92 (92.9%; 95% CI, 87.9-98.0%). The most common causes of SB were placental (n=40 [40.4%; 95% CI, 30.7-