The Impact of Fetal Diagnosis on Duct-Dependent Congenital Heart Lesions

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Introduction

- Fetal diagnosis facilitates perinatal management of infants with congenital heart defects (CHD).
- Previous retrospective studies show that fetal echocardiography accurately detects CHD; however there are mixed results for the benefit of fetal diagnosis
- While antenatal diagnosis is possible for most lesions, its main impact in CHD is to improved morbidity and mortality in neonates with arterial duct-dependent lesions.

Objectives

- We sought to examine acuity of illness on presentation of prospectively enrolled neonates born with duct-dependent CHD.
- Primary outcome was acuity of illness in the neonatal period of infants diagnosed antenatally versus postnatally on presentation to our centre for active management.

Methods

- Nested retrospective cohort study of prospectively enrolled cases with PDA-dependent CHD between January 1, 2009-October 31, 2009.
- Inclusion Criteria:
  - -Fetal or postnatal diagnosis of duct-dependent CHD.
  - -≥36 weeks gestational age and birthweight ≥ 2 kg.
  - -CHD requiring intervention within the neonatal period.
- Exclusion Criteria:
  - -Major extracardiac congenital or genetic abnormalities.
- Primary outcome was initial lactate at initial presentation to the hospital.
- Other outcome measures included:
  - -age at prostaglandin initiation.
  - -highest dose of prostaglandin.
  - -days to intervention.
  - -length of intensive care (ICU) stay pre-intervention.
- Statistical analysis:
  - -Unpaired *t*-test was used to compare the fetal vs. postnatal cohorts
  - -Data are presented as means with SD.

Results

- Of 100 prospectively enrolled neonates, 30 had fetal and 28 had postnatal diagnosis of duct-dependent CHD with active management. The distribution of major lesions is shown in **Figure 1**.
- The 2 study cohorts were comparable in birth weight, gestational age and APGAR scores (**Table 1**).
- As shown in **Table 2**, the fetal cohort had significantly lower lactate levels at presentation, received significantly less likely to be treated with preoperative prostaglandin, were earlier admitted to our centre and underwent surgery earlier.

Conclusions

- ❑ **Facilitated management of prenatally diagnosed duct-dependent CHD was associated with lower lactates, lower prostaglandin dosages, earlier age of transfer to our centre and surgery at an earlier patient age.**
- ❑ **Fetal diagnosis did not impact length of ICU stay prior and after surgery.**
- ❑ **Limitations of our study include the small patient numbers and that we do not account for neonates who may have died prior to transfer to our center because of undiagnosed CHD.**

Figure 1. Distribution of duct-dependent CHD lesions between fetal vs. postnatal cases.

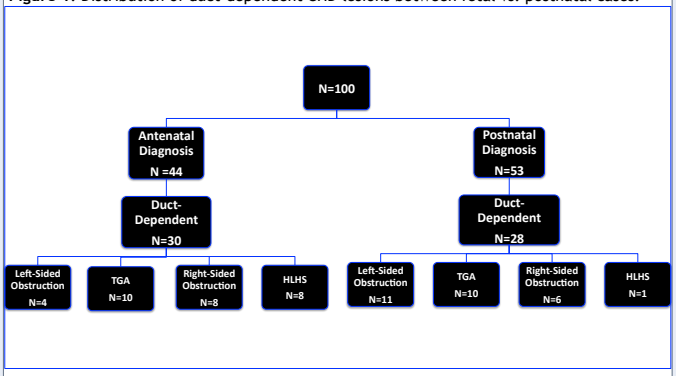

Table 1. Baseline characteristics of enrolled neonates. GA = gestational age

	Fetal Diagnosis N=30	Postnatal Diagnosis N=28	P-value
Male Infants	18/30	15/28	NS
GA at Diagnosis (weeks)	24.9 ± 6.23	39.6±1.6	<0.001
GA at Delivery (weeks)	38.7±1.44	39.2±1.23	NS
Birthweight (kg)	3.35±0.67	3.17±0.52	NS
1 Minute APGAR	8±1	8±2	NS
5 Minute APGAR	8±1	9±1	NS
Neonatal Resuscitation	4/30	2/28	NS

Table 2. Outcomes of neonates with fetal vs. postnatal diagnosis of duct-dependent CHD.

	Fetal Diagnosis N=30	Postnatal Diagnosis N=28	P-value
Age of prostin initiation (days)	1±0	3.52±3.52	NS
Maximal PGE dose (mcg/kg/min)	0.03±0.04	0.06±0.04	<0.05
Age of transfer (days)	1.1±0.6	4.9±6	0.001
Initial pH	7.3±0.09	7.22±0.21	NS
Initial lactate (mmol/L)	3.64±2.04	5.7±4.4	0.04
Initial creatinine (µmol/L)	62.5±12.7	68.3±32.9	NS
Time to surgery (days)	5.5±3.1	8.8±7.1	0.02
ICU stay pre-intervention (days)	3±2.4	2.9±2.7	NS