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Feasibility of Visualizing The Cerebellum at 11-13 Weeks

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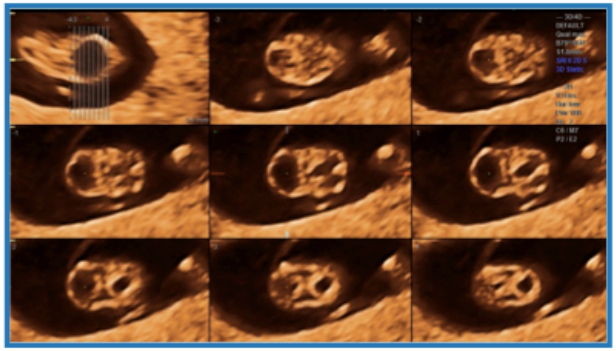
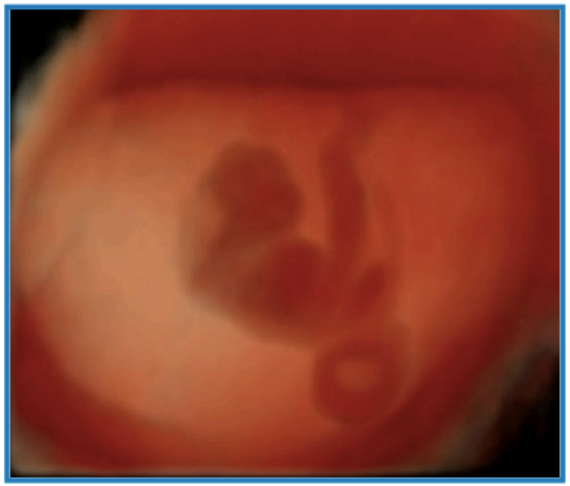
Cerebellum at 11-13 Weeks

Background

- Aristotle (384 BC) “parencephalon” (Clark 1996)
- Described by Vesalius and Willis 1664 *(Ito 2002)*
- Function noted by Rolando 1809 *(Ito 2002)*
- Develops from the rhombencephalon *(Muller 1990)*
- Disorders of vermis or hemispheres *(Ramaekers 1997)*
- Lag in visualization of developed structure *(Babcook 1996)*
- Delay in vermian development *(Bromley 1994)*
- Normogram TCD established by strict criteria *(Egle 2011)*
- IT/BSOB in Spina Bifida *(Chaoui 2009, Lachmann 2011)*
- Limitations in post fossa at 11-13 weeks *(Syngelaki 2011)*



Adapted from "A Practical Guide to 3D Ultrasound"
RS Abu-Rustum. CRC Press 2014



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Cerebellum at 11-13 Weeks

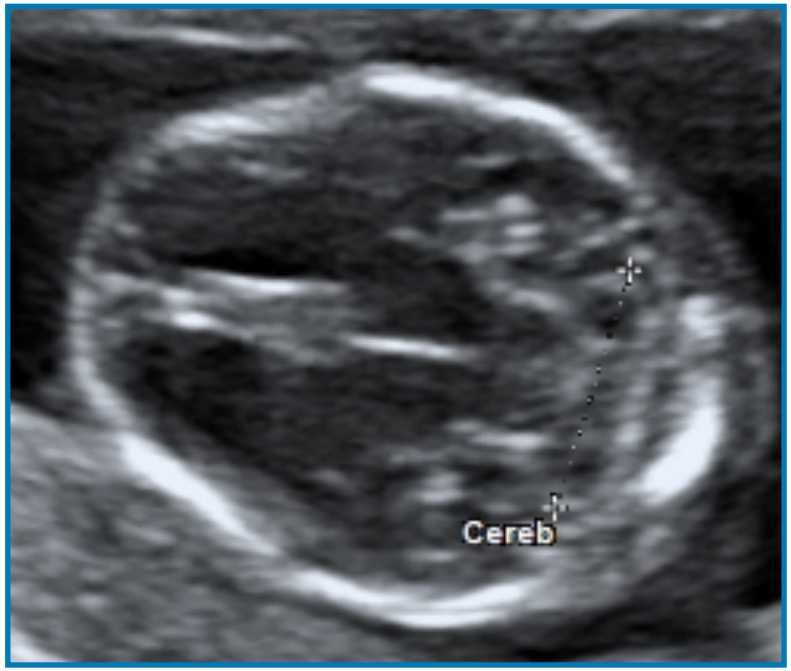
Objective

- Feasibility of visualizing a clear cerebellum at 11-13 wks

Methods

- Prospective transabdominal study
- 139 normal fetuses at 11-13 wks

- Axial plane for BPD and butterfly
- Sagittal plane for NT, IT and fCM
- Axial tilt for the cerebellum
- TCD measured if clear anatomy



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Results

- **Successful visualization in 19/139 (13.7%)**

Maternal and Fetal Characteristics (means and SD)

Variable	Seen 19/139 (13.7%)	Not Seen 120/139 (86.3%)	P-Value
Maternal Age (years)	28.9 (5.54)	29.9 (5.73)	0.481
BMI (kg/m^2)	24.2 (3.50)	24.8 (4.07)	0.555
GA (weeks)	13.2 (0.32)	12.99 (0.51)	0.128
CRL (mm)	74.9 (7.43)	68.3 (6.54)	<0.0001
BPD (mm)	24.4 (1.70)	22.5 (2.26)	<0.0001
NT (mm)	2.14 (0.50)	2.13 (0.49)	0.94
IT (mm)	2.14 (0.49)	2.08 (0.59)	0.663
fCM (mm)	1.56 (0.3)	1.41 (0.57)	0.268

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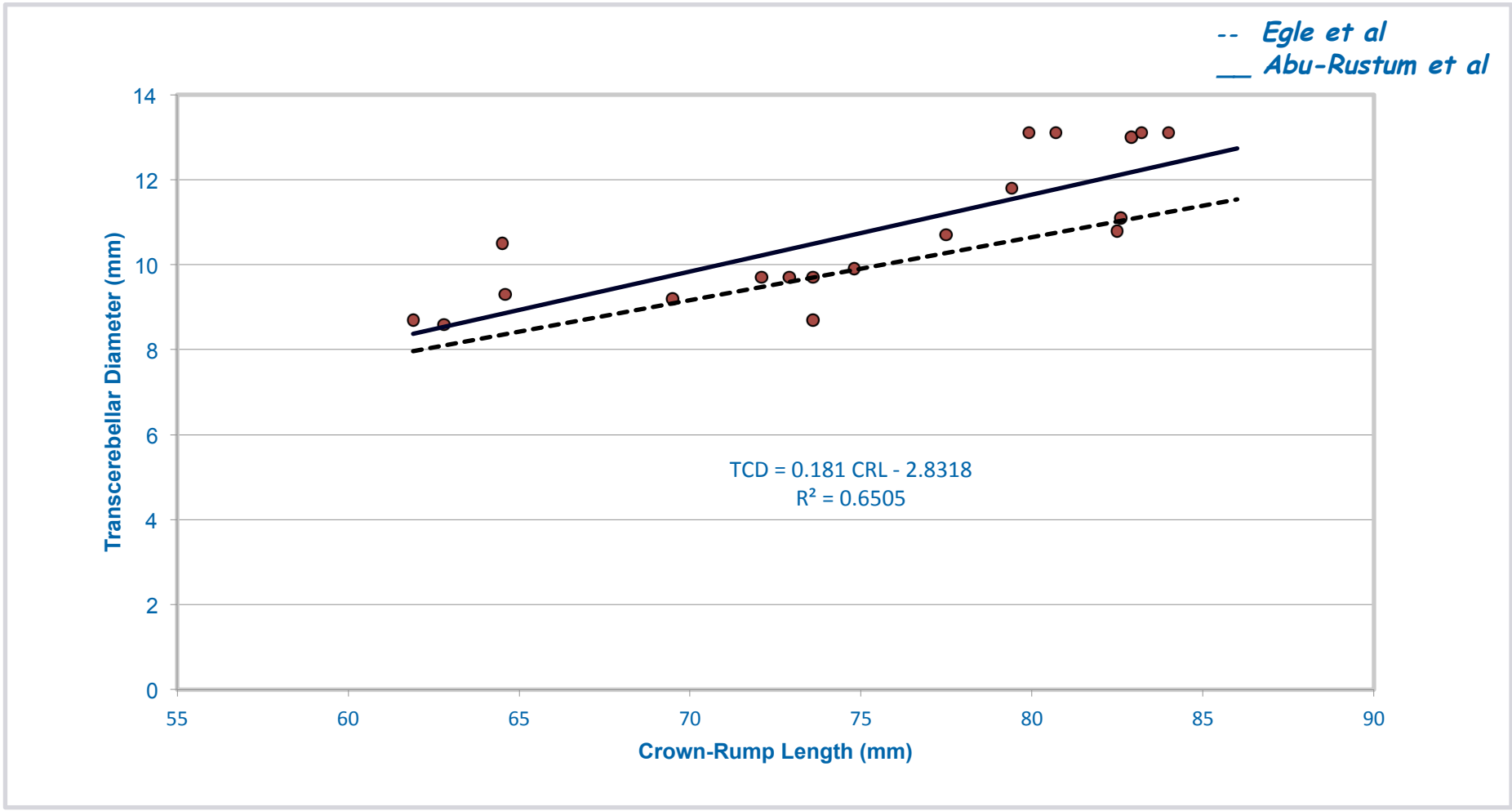
Results



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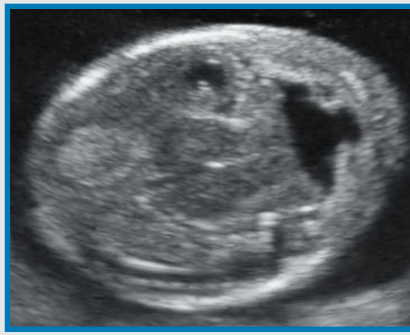
Cerebellum at 11-13 Weeks

Limitations

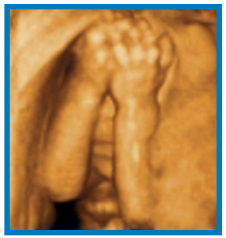
- Small transabdominal study

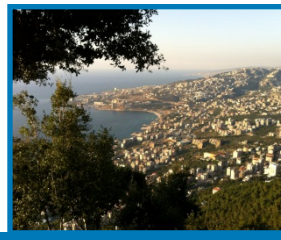
Conclusions

- Adequate visualization in only 13.7% of fetuses at 11-13 weeks
- Fetal CRL and BPD are the most influential factors
- Second trimester evaluation remains a must
- Babcook 1996: “When can we confidently exclude an anomaly?”





Thank you from Lebanon…

Normogram for the First Trimester BPD at 11-14 Weeks in an Unselected Lebanese Population

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Objective

The study's objective was to establish the normogram for the BPD in an unselected Lebanese population and to compare it to the normogram established by Salomon et al in order to determine any ethnic variation.

Methods

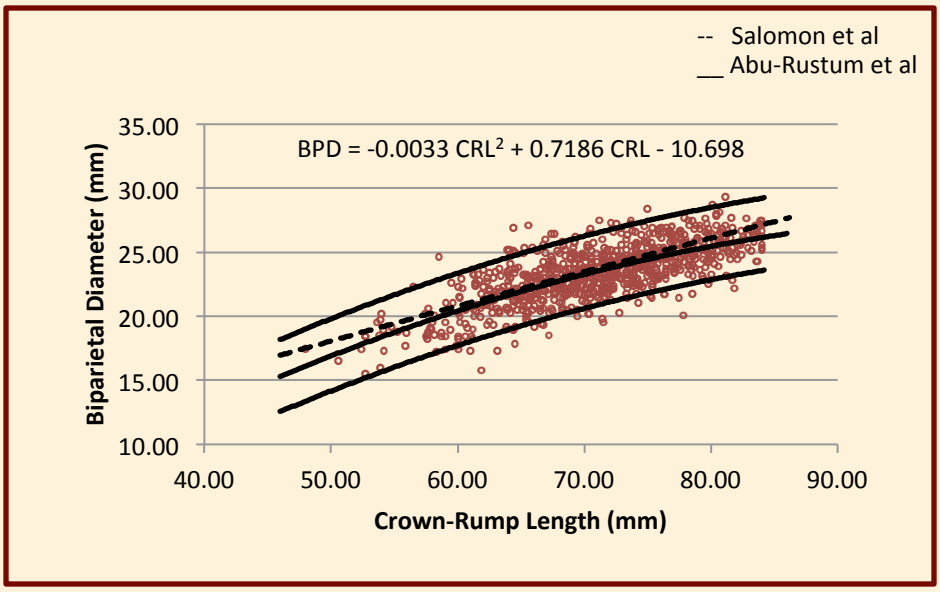
Prospective study on 820 fetuses with confirmed dating undergoing a first trimester scan at 11w6d to 13w6d. The fetal CRL and BPD were measured on all fetuses. Twin gestations, cases with aneuploidy or structural abnormalities and fetuses with an NT > 95th centile were excluded. All fetuses were term live births with a normal neonatal examination at birth. Regression analysis (polynomial second degree) was used to establish the relationship between the fetal BPD and the fetal CRL. P < 0.05 was considered statistically significant.

Results

A total of 820 fetuses were included in the analysis. Mean GA was 13.1 weeks. Mean CRL was 71.1 mm. Mean BPD was 23.4 mm. Mean maternal BMI was 24.95. Our data revealed that the BPD increases with CRL according to the following relationship: BPD = -10.698 + (0.719 × CRL) – (0.33 × 10^{-2} × CRL^2) with a p-value of < 0.0001 and an R^2 of 0.56. There is no statistically significant difference between our normogram and that established by Salomon et al.

Conclusion

Our study demonstrates that in an unselected low risk Lebanese population the normogram for the fetal BPD at 11-14 weeks is similar to what has previously been established by Salomon et al with no evidence for any ethnic variation.



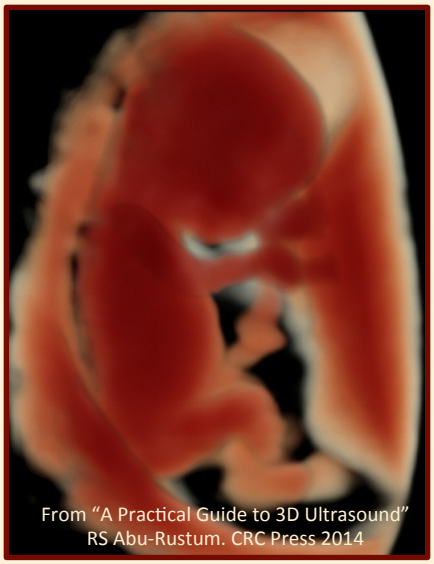
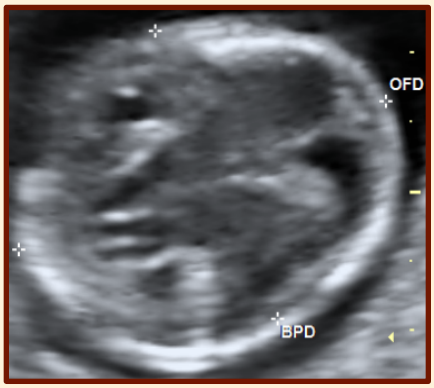


Table 1 Mean, median, range and standard deviation of measurements (*n* = 820)

Variable	Mean	Median	Minimum	Maximum	SD
GA (weeks)	13.1	13.1	11.1	14.1	0.45
CRL (mm)	71.1	71.5	48	84	6.75
BPD (mm)	23.4	23.5	15.5	29.3	2.29

GA, gestational age; CRL, crown-rump length; BPD, biparietal diameter



Is the Brain-Sparing Effect Described in Fetuses with Congenital Heart Disease Apparent in the First Trimester?

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Objective

According to the brain sparing effect described in fetuses with congenital heart disease (CHD), there is a redistribution of cerebral blood flow with cerebral vasodilatation in those fetuses in the face of compromised fetal oxygenation. This compensatory protective mechanism may be insufficient to maintain normal brain growth and development. It has been noted that fetuses with hypoplastic left heart (HLH) and transposition of the great arteries tend to have a smaller head whereas those with coarctation of the aorta (CoA) and tetralogy of Fallot (TOF) tend to have a larger head. As such the aim of this study was to determine whether the brain sparing effect, as reflected by the fetal BPD, may be apparent in the first trimester.

Methods

This was a retrospective study on 820 controls and 32 fetuses known to have congenital heart disease who had undergone first trimester screening for aneuploidy and structural fetal abnormalities at 2 centers in Lebanon. All scans were carried out transabdominally by 2 experienced sonologists. All fetuses had the NT, CRL and BPD measured. Maternal age, BMI, gravidity and parity were obtained. The presence of extracardiac anomalies and aneuploidy in fetuses with CHD was recorded. Live born fetuses were all evaluated by a pediatric cardiologist and the diagnosis confirmed. The BPD as a function of the fetal CRL in fetuses with CHD was plotted against the established normogram. The fetuses with CHD were divided into 3 groups: the fetuses with HLH, the fetuses with CoA and finally the fetuses with other cardiac anomalies.

Results

There were a total of 32 fetuses included in the analysis: 9/32 (28.1%) with HLH, 2/32 (6.3%) with CoA and the remainder, 21/32 (65.6%) with AV canal, HRH, VSD and various other cardiac abnormalities. Karyotype was only available on 6/32 (18.8%) fetuses and of those, 5/6 (83.3%) had trisomy 21 and there was 1/6 (16.6%) with an unbalanced translocation. Extracardiac abnormalities were present in 4/32 (12.5%). Of the fetuses with CHD, 13/32 (40.6%) were live born. Termination of pregnancy was carried out on 14/32 (43.8%). There was spontaneous in utero demise in 2/32 (6.3%). There were 3/32 (9.4%) that were lost to follow up. The fetal BPD as a function of the CRL revealed that there is no difference between fetuses with CHD and normal fetuses.

Conclusion

The brain sparing effect, as exemplified by alterations in the fetal BPD in fetuses with CHD in comparison to normal fetuses, is not apparent in the first trimester.

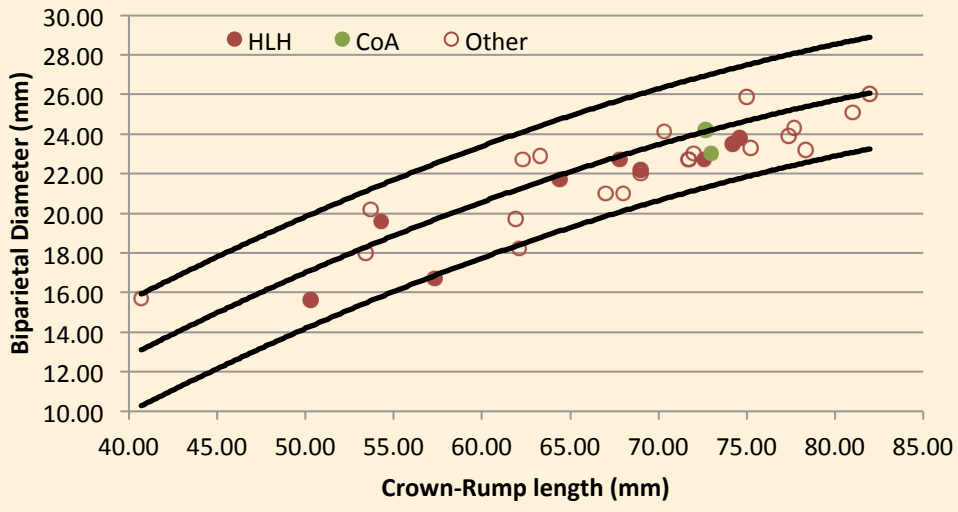
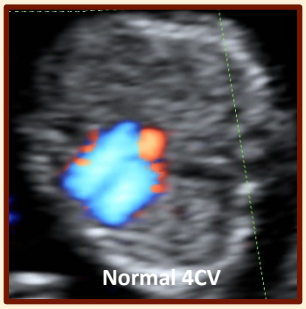
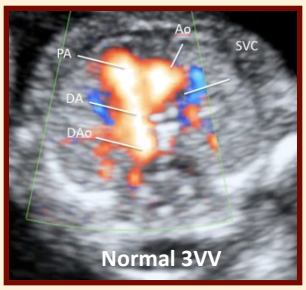
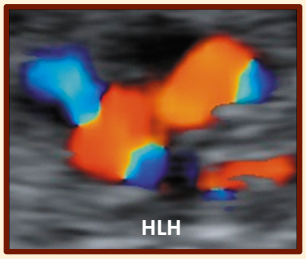


Table 1 Population Characteristics

Variable	Controls (n=820)	HLH (n=9)	CoA (n=2)	Other (n=21)	P-Value
Maternal Age (years)	28.9 (5.13)	30.2 (7.98)	30.0 (-)	29.8 (7.22)	0.718
Body Mass Index (kg/m^2)	24.95 (4.09)	24.6 (3.69)	23.4 (0.99)	24.7 (4.01)	0.942
GA (weeks)	13.1 (0.45)	12.9 (0.88)	13.5 (0.29)	12.9 (0.49)	0.064
CRL (mm)	71.1 (6.75)	64.9 (9.00)	72.9 (0.21)	68.3 (10.23)	0.014
BPD (mm)	23.4 (2.29)	20.9 (2.99)	23.6 (0.85)	22.1 (2.65)	0.001







An Atypical Prenatally-Detected Chest Mass Simulating Postnatal Cardiomegaly

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Case Report

A 29 year old, Garvida 2 Para 1001, BMI 36.6, had had an uncomplicated prenatal course. First trimester scan was normal with an NT of 1.4 mm at 11w5d. Second trimester genetic scan was also normal. During routine third trimester follow up for fetal growth at 34w3d there was appropriate interval growth and a normal amniotic fluid index. However a well circumscribed central to right-sided pericardiac mass was noted measuring 26.5 x 12.1 mm (Figure 1). There was no feeding vessel or any notable flow on color Doppler. It seemed to move with the beating heart. There was no pericardial or pleural effusion. The mass extended from the level of the 4 chamber view to the level of the 3 vessel view in the location of the thymus where it measured 46.1 x 21 mm (Figure 2) which is > 95th centile for the thymus at this gestational age. There was no sign of a congenital diaphragmatic hernia. The heart had normal situs, normal axis, and normal symmetrical 4 chambers with normal outflow tracts. Both atrioventricular and semilunar valves were normal. There was no evidence of any regurgitation. In addition, 2 pulmonary veins were seen entering the left atrium and the sagittal views for the great arteries as well as the bicaval views were all normal. The mass did not have the appearance of neither a rhabdomyoma nor CCAM. The differential diagnosis included pulmonary sequestration, a thymoma or an enlarged thymus. Consultation with pediatric cardiology confirmed the findings. The patient delivered a live born male at 38w1d weighing 3560 grams. Postnatal echocardiography confirmed the prenatal findings of a normal fetal heart and a prominent enlarged thymus which on chest X-ray simulated cardiomegaly (Figure 3).

Our case demonstrates that though it is an unusual prenatal finding, an enlarged thymus may mimic a chest mass. It is more common for it to be diagnosed postnatally by cardiologists to whom neonates are referred for cardiomegaly noted on a postnatal chest X-ray. The enlarged thymus more commonly presents as postnatal pseudo-cardiomegaly. Care must be taken to avoid undue parental anxiety by a thorough evaluation pre- and postnatally.

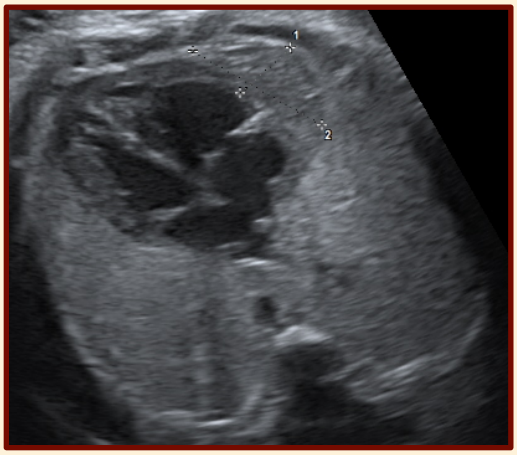

Figure 1

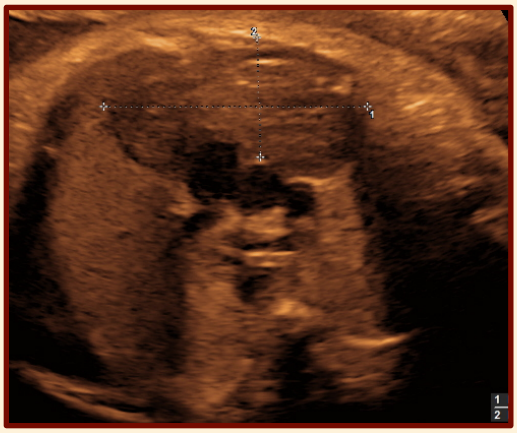

Figure 2

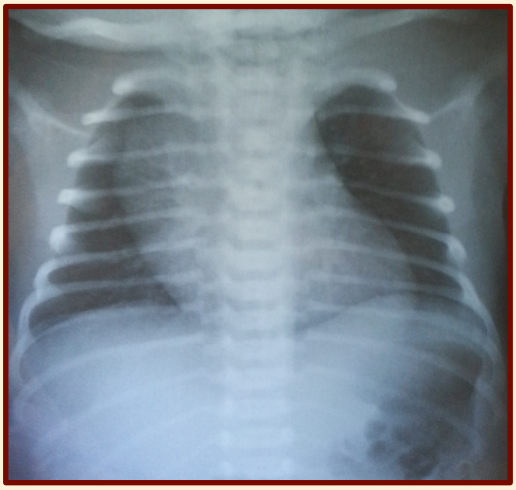

Figure 3