Cardiac Evaluation of the Infant

CHET Education
Dec. 5, 2008
LDF
Congenital Heart Disease

• Most common of all congenital birth defect
• Affects 8 per 1000 live births
• Prompt recognition, stabilization and referral are crucial to outcome
• Due to the complexity of this patient population these kids are most challenging even to the seasoned practitioner
Pulmonary vs. Cardiac

• Pulmonary
  – No murmur
  – + $O_2$ challenge (inc Sat > 10% with 100%)
  – Changes in PCO$_2$ (inc or dec)
  – CXR – small heart, parenchymal changes, atelectasis, hyperinflation, etc
  – Quiet precordium
  – Increased respiratory distress, tachypnea, retractions, obvious inc in WOB
Cardiac vs. Pulmonary

• Cardiac
  – +/- murmur
  – - O₂ challenge (< 10% change in Sat in 100%)
  – Quiet tachypnea
  – Minimal changes in PCO₂
  – CXR - +/- large heart/ pulmonary edema
  – Palpation of active precordium
  – More cyanosis with agitation (shunt)
Cardiac Presentation

• Three typical presentations depending on lesion
  – Cyanosis
  – Cardiovascular collapse
  – Congestive heart failure/pulmonary overcirculation
Cyanotic Lesions

- "5 Ts"
- Transposition
- Tetrology of Fallot with pulm atresia/ stenosis
- Tricuspid atresia
- Truncus arteriosus
- Total anomalous pulmonary venous return (obstructed)
TGA

• Most common cyanotic lesion in **newborn period** 5% of all CHD
• 90% present in first day of life
• Ao arises from RV; PA from LV; **parallel circ**
• Must have mixing at Atr/Vent or ductal level
• CXR oval hrt due to anterior Ao and large RV
• PGE$_1$ life saving to maintain pulm blood flow
• Definitive repair – Switch and septal defect closure
TET

- 6-10% of congenital heart disease, most common
- Constellation of large VSD, RVOT obst, overriding Ao, RVH
- Degree of cyanosis dependent on RVOT obst
- The more severe the obstruction the earlier the presentation
- Mild obstruction presents later/ “pink” TET
• CXR – boot shape 2\textsuperscript{nd} to RVH
• Severe TET with pulm atresia requires PGE\textsubscript{1} to maintain pulm blood flow (acutely)
• Definitive repair now done as a single stage primary repair (vs. BT shunt then repair )
Tricuspid Atresia

- 1% of all CHD, 3rd most common
- No direct connection of RA to RV
- Must have ASD and shunts R > L
- RV is underdeveloped
- Ductal dependent/ PGE$_1$ to maintain pulm blood flow
- Definitive repair BDG -> Fontan
TA
Truncus Arteriosis

- Less than 1% of all CHD
- Single great artery supplies systemic, pulmonary and coronary circulation
- Degree of cyanosis depends on amount of pulmonary blood flow
- **Classically grouped with cyanotic lesions, also pulm overcirculation with some degree of CHF**
- Can present with tachypnea, cardiomegaly, + murmur, bounding pulses 2\textsuperscript{nd} to pulmonary run off
Truncus

• OR cyanotic from R -> L shunt 2\textsuperscript{nd} to respiratory disease/infection (parallel circulation)
• Early surgical intervention with RV to PA conduit
Truncus
Total Anomalous Pulmonary Venous Return

- Pulmonary veins drain into systemic venous circulation
- Supracardiac, cardiac, infracardiac (SVC, RA, or portal circulation)
- Systemic circulation maintained by R -> L shunt at atrial level (ASD)
- Obstruction most often with infradiaphragmatic connection
TAPVR

• Significant obstruction presents with cyanosis and pulm edema

• Partial or unobstructed will present later, generally within the first 2 months with hx of poor feeding, tachypnea, wheezing and mild cyanosis
TAPVR - infracardiac
Cardiovascular Collapse

- First 2 weeks of life
- Medical emergency
- Infants are discharged prior to the ductus closing
- Present in shock
- PGE$_1$ life saving
CV Collapse - Lesions

- Hypoplastic left heart syndrome
- Critical aortic stenosis
- Coarctation of the Ao and interrupted aortic arch
HLHS

- Most common cause of death during first week of life
- Diminutive LV, critical AS, mitral stenosis, hypoplastic proximal aorta
- Ductus closes, PVR reduced
- Shock ensues rapidly to decrease systemic flow
- \( \text{PGE}_1 \), maneuvers to increase PVR (parallel circ)
- Three definitive options: transplant, Norwood, nothing
HLHS
Critical AS

- Shock 2\textsuperscript{nd} to compromised systemic flow
- Ductus closes
- PGE\textsubscript{1} again is life saving
- Airway support, inotropes, treat acidosis
- If LV too small may need Norwood
- If LV adequate, valvotomy or balloon valvuloplasty in cath lab
Coarc/IAA

• Coarc 5-8% of CHD, IAA < 1%
• Ductus closes, severe shock
• Discrepancy of upper and lower pulses and BP
• PGE$_1$, airway support, treat acidosis, inotropes for severe early presentation
• Milder forms may present later but still with significant shock
Congestive Heart Failure

• Presents more gradually, beyond neonatal period
• Symptoms vague – poor feeding, wt. gain etc.
• + murmurs usually appreciated
• Two groups – pulmonary overcirculation, most common
• Ventricular failure, less common
Pulm Overcirculation

- Mod to large VSD
- AVC or AV septal defect
- Large PDA
Overcirculation

- Signs of CHF
- PVR falls, pulmonary blood flow increases
- + murmur
- CXR – large heart, pulm edema
- Airway support, diuretics, afterload reduction surgical repair
PDA
Ventricular Failure

- Anomalous left coronary artery
- Myocarditis
- Cardiomyopathies
Anomalous Left

- Coronary Artery originates from the pulmonary artery
- PVR falls
- Steal as blood actually flows retrograde from coronary circulation to pulmonary circ
- Severe shock, ischemic myocardium
- Increase PVR, volume to perfuse heart
Myocarditis/Cardiomyopathy

- Poor feeding, pallor, tachypnea, sweating
- Shock
- Hepatomegaly
- Cardiomegaly, increased pulmonary markings
- Milder inotrope, afterload reduction, diuresis
- Severe need intubation
Cardiomyopathy
END