B D R News

The official newsletter of The Birth Defects Registry of India, (A Unit of Fetal Care Research Foundation)

Volume 4 Issue 3: Oct 2004

PROCEEDINGS OF THE BIRTH DEFECTS REGISTRY MEETING HELD ON 16/10/04

The final quarterly meeting of the Birth Defects Registry of India for the current year was held on the 16th of October 04. CME on Congenital Cardiac Defects was held on that day. Two dignitaries from Frontier Life Line Hospital, Chennai had delivered lectures. Dr.Premsekar, Consultant Pediatric Cardiologist spoke on the "Medical Management of Common Congenital Cardiac Anomalies"and Dr. Ravi Agarwal, Consultant Pediatric Cardiac Surgeon spoke on the "Surgical Management of Common Congenital Cardiac Anomalies". At the commencement of the meeting Dr. S. Suresh welcomed the gathering. He mentioned that application for membership in the International Clearing House for Birth defects-Rome has been forwarded as a follow up of the last meeting. He also invited interesting case presentations from all the members for the CME on Birth defects to be held in January next year.

This was followed by a short inaugural proceeding of the newly formed Nagercoil BDR represented by the Nodal member Dr. Sashya Jayaharan, Consultant Paediatrician, Dr. Jayaharan Memorial Hospital, Nagercoil. BDRI plaque and BDRI Starter Kit were presented by Dr. Indrani Suresh.

Dr. Premsekar lauded the efforts of Dr. Suresh to find out the prevalence of birth defects in our population. He said that it is a difficult venture in the present scenario keeping in mind the size of the population and the existing data systems in our country. He pledged his support for the cause of the organization. Excerpts of the guest lectures are given below:

Medical Management of common congenital heart defects
Dr. Premsekar, Frontier Life Line Hospital, Chennai

Dr.Premsekar began his lecture with the neonatal presentation of congenital heart defects. When a neonate presents with respiratory distress and cyanosis, the neonatologist is faced with the daunting task of identifying the etiology for the symptoms to implement specific management .

Cyanosis: Cyanosis may be due to neurological, pulmonary or cardiac etiology.

• The newborn with cyanosis of neurological etiology typically is a hypotonic baby with shallow irregular respirations with the cyanosis resolving on external stimulation of the baby. The cause is usually perinatal asphyxia, hypoglycaemia, excessive maternal sedation, and intracranial haemorrhage.

• Babies with cyanosis of pulmonary etiology typically exhibit significant tachypnoea with grunting respirations. The cause could be one of the many pulmonary parenchymal diseases or pulmonary vascular disease. Here again the cyanosis decreases significantly with oxygen administration.

• In newborns with cyanotic congenital heart diseases there is central cyanosis which does not resolve with oxygen administration. This forms the basis for the hyperoxia test.

Hyperoxia Test is done by administering 100% oxygen to the baby for 10 minutes followed by an arterial blood gas analysis. If the PO_2 exceeds 250, the baby is said to have passed the hyperoxia test indicating that the baby does not have a cyanotic heart disease. If the Po_2 falls below 160 the baby has failed the hyperoxia test indicating the presence of a cyanotic cardiac problem. A PO_2 exceeding 160 would rule out a cyanotic cardiac problem but not conclusively. The baby may or may not have associated shock depending on the adequacy of the ductal flow on which either the pulmonary or systemic circulation is dependant. A baby presenting with cyanosis and shock and additionally failing the hyperoxia test warrants an immediate cardiac evaluation. Only echocardiography can definitively detect the underlying lesion. This may not always be available and based on suspicion of cardiac pathology, measures need to be taken to stabilise the baby.

Stabilisation of the baby:

• **Temperature** - Maintenance of a neutral thermal environment to minimise the O_2 requirement.

• **Intravenous fluids** - A fluid bolus of 10-20 ml/kg over 15 minutes enhances the ductal flow and augments the cardiac output by increasing the post ductal circulation. If not in shock and if there is clinical and radiological evidence of pulmonary congestion, maintenance fluids at 100 ml/kg/day or 75 ml/kg/day should be started.

• **Oxygen administration & Ventilatory Support** - Administration of oxygen can be continued as there may be an element of pulmonary pathology accounting for the cyanosis and the adverse effects of oxygen administration in a baby with a cyanotic problem overweighs the advantage provided by it in a sick, cyanotic neonate of doubtful etiology. Ventilatory support would help reduce the energy expenditure and oxygen demand. The peak end expiratory pressure can be modified according to the underlying pathology; high when there is pulmonary congestion and low in conditions with reduced pulmonary flow.

• **Nutrition** - A total calorie intake of 120 – 140 Cal/kg/day is aimed at.

• **Diuretic therapy** – Frusemide at 1 mg/kg/dose is required, when there is pulmonary and right heart congestion.

• **Inotropic support** - In acutely sick neonates, dopamine infusion at 5-15 mcg/kg/min (30 mg/kg in 50 ml of normal saline and infusing at 1ml/hr delivers the drug at 10 mcg/kg/min.) should be given.

• Prostaglandin infusion at 0.01 – 0.1mcg/kg/min is needed when there is a strong suspicion of a duct dependant circulation as shown by a failed hyperoxia test. This is justified even in the absence of a definitive diagnosis by echocardiography as ensuring ductal patency in a duct dependant condition would be life saving. The prostaglandin infusion of 0.01 mcg/kg/min is worked out by adding 30 mcg/kg of prostaglandin E1 to 50 ml of normal saline and infusing at 1ml/hour. The infusion can be titrated depending on the clinical response. The two main side effects of prostaglandin, namely hypotension and apnoeas need to be taken in to account and it is advisable when transferring a baby on prostaglandin infusion to do so with the baby on ventilatory support.

Classification of Neonatal cardiac problems
- Cyanotic
- Acyanotic.

The acyanotic group can be further classified in to conditions with: • Left to right shunts –Ventricular septal defect (VSD), Patent ductus arteriosus (PDA), Aorto-pulmonary window, Aorto-ventricular tunnel and Atrial septal defect(ASD). Depending on the size of the defect and the degree of the left to right shunt, the baby exhibits tachypnoea, intercostal recessions and poor suck. Apart from the atrial septal defect, the rest of the lesions are high velocity, systemic to pulmonary shunts and therefore are more likely to make the baby symptomatic.

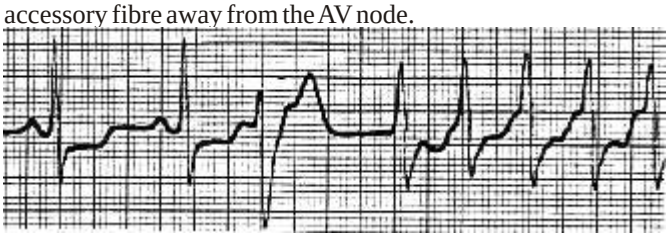
• Obstructive pathology - Pulmonary valve stenosis, Aortic valve stenosis, Coarctation of aorta(COA), Obstructed cor-triatrium, and rarely Pulmonary vein stenosis. Again the symptoms depend on the severity of the obstructive condition and as a general rule, left sided obstructive lesions tend to be more symptomatic than the right. When symptomatic, these conditions warrant mechanical relief of the obstruction either in the form of ballooning or when this is not feasible, surgical intervention.

• Regurgitant valve lesions are unusual at birth and severe mitral valve regurgitation may produce symptoms of breathlessness and tachypnoea owing to the pulmonary venous congestion.

The Cyanotic group can be broadly classified in to the following conditions with: • Decreased pulmonary flow - Tetralogy of Fallot (TOF), Double outlet right ventricle(DORV)with Pulmonary Stenosis(PS), Pulmonary Atresia with intact ventricular septum, Transposition of great arteries(TGA) with VSD and PS, Tricuspid Atresia with PS and Hypoplastic right ventricle. When these conditions are associated with very severe right ventricular obstruction or atresia then the ductus remains the only channel for pulmonary circulation.
• Increased pulmonary flow – DORV with unrestrictive pulmonary flow, Tricuspid Atresia, Truncus arteriosus, AV canal defect, TGA with VSD and PDA and Univentricular heart with unrestrictive pulmonary flow.
Pulmonary venous congestion - Obstructed Total anamolous pulmonary venous drainage, Hypoplastic left heart with Mitral Atresia and restrictive ASD.

Arrhythmias

Apart from the congenital cardiac structural lesions, the other cardiac problem that neonates present with is arrythmia - the most common being atrioventricular re-entrant tachycardias. For re-entry to occur there must be atleast two pathways (A or alpha pathway and B or beta pathway), with unidirectional absence of conduction in one and slowed conduction in the other. The differing electrophysiologic characteristics of the two pathways allow re-entrant tachycardias to develop. The trigger for the re-entry is usually an atrial or a ventricular premature beat. When this process leads to a single activation, the result is just one premature beat, whereas repetitive reactivation results in a sustained tachycardia. The characteristics of re-entrant tachycardias are that they are usually paroxysmal, have a fairly constant rate and can be induced or terminated by pacing. It is characterised by a paroxysmal onset and termination. The typical heart rate is above 220 /min and the ECG shows a narrow QRS complex tachycardia with no preceding P wave. The P wave may be either buried within the QRS complex as in nodal re-entrant tachycardias (when both limbs of the re-entrant pathway is within the AV node) or it may be seen following the QRS complex in the ST segment when the re-entrant pathway is an accessory fibre away from the AV node.



Acute management:

1. IV access

2. SVT to be established - Narrow complex tachycardias at > 220 /min, P wave invisible or seen following the QRS complex.

3. IV Adenosine fast bolus injection – should be started with 50mcg/kg/dose increasing to 100 & 200 mcg/kg/dose until SVT is interrupted with 2 minutes interval between the injections.

4. If not responding to adenosine, the child may be sedated and DC shock at 2 J/kg can be given.

Long term management:

1. Medical (Digoxin, Propranalol, Flecainide as suited for each patient)
2. Electrophysiological Ablation

Congenital Complete Heart Block is yet another arrhythmia which paediatricians and neonatologists may face during their practice. This is recognised by complete disassociation between the P wave and the QRS complex with associated bradycardia. If the baby is symptomatic with hypotension secondary to the bradycardia, management is as follows:

Management :

Isoprenaline infusion 0.1 – 2.0 mcg/kg/min. (< 33 kg: 0.3 mg/kg in 50 ml at 1 ml/hr = 0.1 mcg/kg/min.) may be given during acute crisis. Permanent pacemaker is the option for long term management.

SURGICAL MANAGEMENT OF COMMON CONGENITAL CARDIAC ANOMALIES –
Dr. Ravi Agarwal, Frontier Life Line Hospital.

Dr. Ravi Agarwal's presentation on the surgical management of common congenital cardiac defects was well depicted with candid illustrations. He said that Congenital Heart Defects (CHD) is one of the most common birth defects with an incidence rate between 4-12 / 1000 live births estimated through various hospitals based studies. Approximately 1,12,000 infants with CHD are added to our population every year, Almost 45% of them (i.e. 50,000) require some intervention or surgery during the first year of life (Burden of Rheumatic & Congenital Heart Diseases in India—Indian Heart Journal 2002,54,104-107).

There are two types of cardiac surgeries namely - open heart surgery which is performed on cardio-pulmonary by pass using heart lung machine and it does not involve opening of the cardiac chamber as the name denotes. Cardiac problems like ASD, VSD and TOF are corrected using this technique.
The other type is closed heart surgery where the surgery is performed without using heart lung machine and with the heart beating. This may require opening up of the heart chamber as in PDA ligation and repair of COA.

The Surgeon dealt in detail about the surgical options available, the timing of surgery and the methods adopted in corrective surgeries of a number of cardiac defects. The timing of surgery depends on:

1. The defect per se and the knowledge of its evolution if untreated
2. The size of the defect
3. Associated cardiac and extra cardiac anomalies
4. Effect on the patient - episodes of Congestive Cardiac Failure (CCF), cyanotic spells etc
5. Development of extra cardiac complications particularly on the pulmonary vasculature.

PATENT DUCTUS ARTERIOSUS (PDA):

Anatomic existence of a PDA regardless of its size is an indication for surgery. Timing depends on the severity of the effects on the child. In infants with CHF, pulmonary hypertension hypertension or recurrent pneumonia, surgery is performed on an urgent basis. Correction is done by double ligation and division through left posterolateral thoracotomy without cardio pulmonary bypass or through transpulmonary closure incase of caicified PDA. The presence of pulmonary vascular obstructive disease (PVOD) is a contraindication for surgery.

COARCTATION OF AORTA (COA):

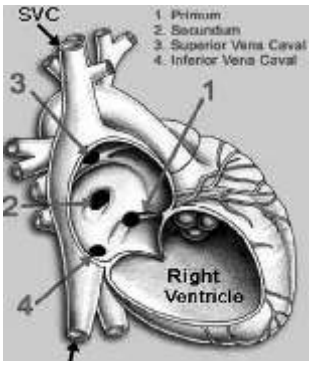
This may be rectified by 3 methods namely

a) Resection and end to end anastomosis, a process consisting of resecting the coarct segment and anastomising the proximal and distal aortas.
b) Subclavian aortoplasty
c) Goretex patch aortoplasty

Post operative follow up every 6-12 months to check for recurrence of COA is necessary if surgery has been performed in the first year of life. Repair could be done in infancy itself if CHF develops early in life or it can be postponed to 3 - 6 months of life.

ATRIAL SEPTAL DEFECT (ASD):

This repair could be performed during pre school age because the possibility of spontaneous closure exists. However surgery is performed if the defect is large leading to complications like CHF which does not respond to medication or if it is associated with broncho pulmonary dysplasia.



The defect is repaired under cardio pulmonary bypass with either a simple suture or a pericardial patch. Right posterior thoracotomy is opted for female children for cosmetic purposes.

VENTRICULAR SEPTAL DEFECT (VSD):

The location, number and the size of the VSDs determine the timing of surgery, Spontaneous closure occurs in 30—40% of patients with membranous and muscular VSD during the first 6 months of life. These do not increase in size with age. Inlet defects and infundibular defects neither reduce in size nor close by themselves.Direct closure of the defect under cardio pulmonary by pass and /or deep hypothermia using a Goretex patch is preferably carried out through an atrial approach. Pulmonary artery (PA) banding is done if there are muscular and multiple VSDs and if complete repair becomes difficult.. VSD repair may be done within 3 months of life in the presence of intractable heart failure and respiratory symptoms. Primary repair is indicated for children between 3 -12 months according to symptomatic status. In older patients with large VSD a complete evaluation of cardio pulmonary status has to be performed by cardiac cath to decide on the suitability of closure.

ATRIO VENTRICULAR CANAL DEFECT (AVSD)

Surgery is indicated for AVSD during the first 3-4 months of life or earlier if severe heart failure persists. By 1 year of age severe pulmonary vascular disease may set in otherwise. Surgery involves ASD, VSD closure and AV valve repair.

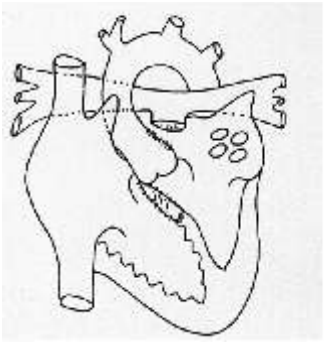
TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION (TAPVC):

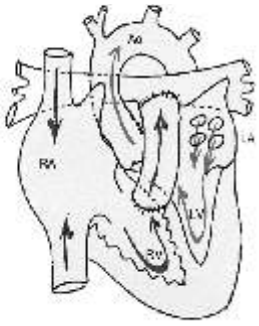
Depending on the drainage site of the pulmonary veins, the defect may be divided into 4 types 1) Supra cardiac 2) Cardiac 3) Infra cardiac 4) and mixed type. Clinical manifestations differ depending on the presence of obstruction to the pulmonary venous return. Immediate corrective operation in neonatal period or infancy is necessary as soon as it is diagnosed. Repair involves anastomosis of the common venous chamber into left atrium. Surgery may be preformed under cardiopulmonary bypass, profound hypothermia and total circulatory arrest.

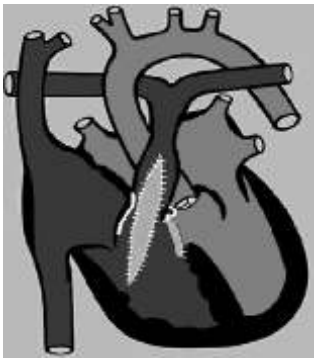
TRUNCUS ARTERIOSUS (TA):

There are 3 types of TA . Diagnosis of TA calls for early repair. 50% of unoperated children die within one month of life.

TRUNCUS ARTERIOUSUS: REPAIR





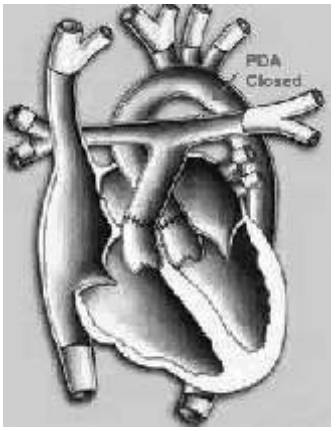


TETROLOGY OF FALLOTS (TOF):

Corrective surgery in this condition involves VSD closure and enlargement of right ventricular outflow tract (RVOT). In severely cyanotic infants shunting on palliative basis is done within 1-2 months of life. Classic Blalock -Taussig shunt anastomosed between the subclavian artery and the ipsilateral PA may be the procedure of choice for infants older than 3 months. Goretex interposition shunt placed in between the subclavian artery and the ipsilateral PA is the procedure of choice for smaller infants less than 3 months old. Pulmonary atresia can be repaired by using homograft and connect the PA to RV.

TRANS POSITION OF GREAT ARTERIES (TGA):

Arterial Switch Operation is the corrective surgery for this condition. Illustration given here clearly explains the surgical procedure involved. When there is TGA and intact ventricular septum with severe cyanosis, Balloon Atrial Septostomy (BAS) is done first followed by Arterial Switch Operation (ASO) within 7 - 10 days of life. If there is TGA and VSD, ASO is performed within first few weeks of life.



TRICUSPID ATRESIA & SINGLE VENTRICLE PHYSIOLOGY

In neonates with tricuspid atresia, palliation is done initially by administering Prostoglandin E to maintain the patency of the ductus before cardiac surgery. Most patients who have atresia with decreased pulmonary blood flow (PBF) need a procedure like Cavopulmonary shunt to increase PBF and improve arterial saturation. If there is increased PBF, PA banding is resorted to.

SINGLE VENTRICULE REPAIR:

In older infants of 6 months age, an end to side SVC to RPA shunt—bidirectional superior cavopulmonary shunt or bidirectional Glenn procedure can be performed. The IVC blood still bypasses the lungs. This is widely accepted palliative procedure that satisfactorily increases oxygen saturation which averages to 85% without adding load to the left ventricle. This is the first of the two stage Fontan-type operation. Completion of this surgery can be done at 5—6 years of age.

COMPLETE CONGENITAL HEART BLOCK(CCHB):

Dr.Ravi Agarwal went on to explain a case with CCHB where a pacemaker was implanted in a 3 days old baby with successful outcome. He concluded his presentation with the following message:

• In the present era of technological advancement, age & weight of the patient are no longer a limiting factor for safe and successful intra—cardiac repair at young age.

• The trend is towards early repair after the diagnosis to avoid the secondary effects of the cardiac lesion on other organs of the body.

During discussion there were enlightening interactions between the audience and the speakers. Some of the issues discussed on the topics are as follow:

1. What is the corrective procedure if TGA is diagnosed late?
If TGA is diagnosed late, Atrial switch operation such as Mustard operation is done to correct the disorder. This oldest form of surgical technique redirects the pulmonary and systemic venous return at the atrial level by using either a pericardial or prosthetic baffle.

2.What is the prognosis of AVSD corrections?

The response here is quite good. It could be done at one stage within 3 months of life as is done in Down children who usually present this problem. They do well postoperatively. In case there is unbalanced ventricles, the surgeries could be done at intervals. Here the prognosis will depend on the severity of pulmonary hypertension.

3.Is there a possibility to miss out Tricuspid Valve Atresia in antenatal scan?

While answering this question, Dr. Indrani Suresh said that targeted scan done around 20 weeks should incorporate 4 chamber and outflow tract examination of the heart. The fetus has to be systematically imaged as one would examine an infant clinically. Sometimes there are chances to miss out the evolving anomalies like pulmonary atresia in TOF during this period. The size of RV provides a clue to the Sonologist about the problem.

4.How often a pace maker has to be replaced?
Pacemakers need to be replaced once in 4/5 years.

5.Should all high risk mothers(eg Diabetic mothers) need to undergo fetal ECHO antenatally?

High risk mothers need to undergo a thorough fetal heart scan by proficient Sonologist at 20 weeks.

6. Do all babies born to diabetic mothers need to have neonatal ECHO?

These neonates need not have ECHO unless they are symptomatic.

7.What is the prognosis of HLHS?

Il involves complicated multistaged surgery and hence mortality rate is quite high here(40%) when compared to VSD.

Support Group for MPS: Activities Update

MPS Children enrolled in the support group had an opportunity of getting consultation from a metabolic specialist on 15th & 16th of September 04. Dr.Ashok Vellodi, Consultant Paediatrician, Great Ormand Street Hospital, U.K examined the children at the special clinic organised by the Fetal Care Research Foundation on these two days. His valuable suggestions are being followed up through a group of good hearted medical specialist based in various institutions at Chennai. The specialists team consisting of medicos, psychologist, physiotherapist & yoga therapist had also attended on the children and given advice during the Multispeciality Clinic held in August this year. Efforts are on to follow up their prescription and to send blood/ urine samples of the children abroad to confirm the diagnosis and type of MPS in those whom it is not confirmed.







An Appeal

If you come across a case of MPS in your practice, kindly enroll him / her in the support group for the benefit of the child.

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NETWORK OF NODAL CENTRES
BIRTH DEFECTS REGISTRY OF INDIA (BDRI)

We are growing...

BDRI is happy to announce that it has been expanding rather rapidly in the past six months. We thank all the Nodal members for their motivation and zealous efforts, without which this would not be possible Dr.T.Mahesh Gandhi,Consultant Obstetrician and Gynaecologist, Jivanyog Nursing Home, Visnagar, North Gujarat opened Visnagar BDR in May 04. Two more registries were commissioned by Dr. Hema Divakar, Consultant Obstetrician and Gynaecologist, Divakars Hospital, Bangalore and Dr. S.Vivekanandan, Consultant Radiologist, Sri Vari Scan Centre, Coimbatore, at Bangalore and Coimbatore respectively in July 04. Dr. Gio Gnanadurai, Managing Director of Gnanadurai Hospital, Sivakasi joined the mission in August 04. While appreciating the commitment and co-operation of the existing members, BDRI also requests their continued support and participation in the future.

Given below are the two interesting case presentations on birth defects, postnatally managed with successful outcome. These were presented by Dr. Narendra (Divakars Hospital) at the inaugural session of Bangalore BDR, organized by the Society for Fetal Medicine & Genetics (SOFEM) and the Bangalore Society of Obstetricians & Gynaecologists (BSOG) on the 4th of July 04. at Divakars Hospital, Bangalore. The team of Paediatric Surgery led by Dr. Ramesh at the Sagar Apollo Hospital, Bangalore performed these surgeries.

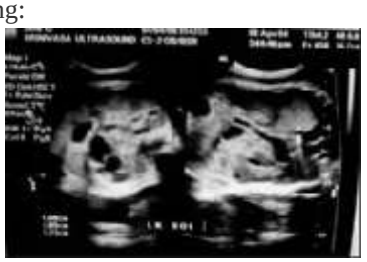
Fetal Supra-Renal Mass - Diagnosis and Implications.

Dr. Hema Divakar, Dr. Chitra Ganesh, Dr. Ramesh. Divakars Hospital, Bangalore

Case - 1

Mrs. B.V, 29yrs old G3P1A1 with an uneventful antenatal period was referred for a mid pregnancy scan. Biometry, structural survey, liqour and placental position were reported normal. Repeat scan at 38 ½ weeks revealed the following:

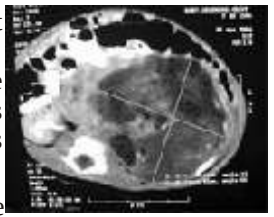
Fetal left renal region showed a well defined ovoid, solid and cystic space occupying lesion measuring 68mm x 48mm x 52 mm. Fetal kidneys, bladder and liquor were normal and there were no other anomalies.



Differential diagnosis of neuroblastoma / teratoma / extralobar pulmonary sequestration was considered.

A female baby weighing 3.75 kg with good Apgar, was vaginally delivered. A Paediatric Surgeon was called to evaluate the child.

• On clinical examination there was an irregular, ballotable mass , palpable in the left lumbar region.

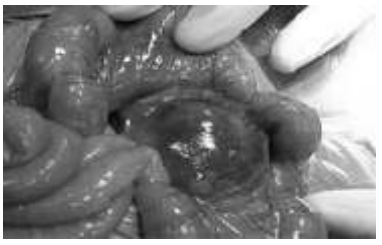


• CT Scan of the abdomen showed a large sized 80mm x 70mm x 80mm soft tissue mass with calcifications in the left suprarenal fossa. It was diagnosed as Neuroblastoma mass extended across the midline anterior to aorta. Left kidney was displaced into iliac fossa and it appeared normal, with no involvement.

•Alpha feto protein (α FP), Human Chronic Gonadotrophin (HCG), Vanillylmandelic Acid (VMA) were assessed to rule out the involvement of adrenal glands. VMA and Homo Vanillic Acid (HVA) are known to increase in adrenal cystic masses secondary to hemorrhage.

• Laprotomy with excision of tumor was planned on D3 and the mass along with left kidney had to be removed, as the renal blood vessels were traversing the mass.

• **Gross Appearance** of the encapsulated globular mass, measuring 80mm x 65mm x 50mm had firm to cystic texture and the cut section revealed brownish, yellowish, cystic areas with cartilage and bone. Soft areas showed jelly like material.



Microscopic Examination showed immature neuro epithelial element and glial tissue (> 75%). Adrenal gland was embedded into this tissue. Papillary structures, glial tissue, internal glands, cartilage, bone, fat, muscle and sweat gland ducts were also seen.

Final Impression of Immature Teratoma - (retroperitoneal) was made. Aggressive treatment with surgical removal present excellent outcomes as in the present case. As this mass was large, observation for spontaneous regression was not favoured.

Conclusion: Prenatal diagnosis of fetal intra abdominal masses is fairly easy and does cause an overwhelming anxiety to the Sonologist, Parents and the Obstetrician. Fortunately, majority of these cystic and solid masses are treatable and recurrent risk is almost unknown.

If there are no associated anomalies or hydrops, the survival rate after surgical intervention is very good. Prenatal diagnosis offers an opportunity for early intervention with excellent prognosis.

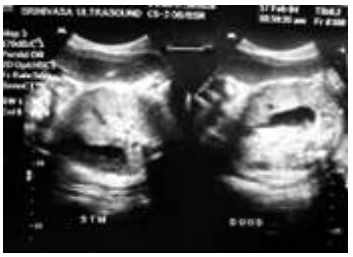
Bowel Obstruction - An unusual antenatal presentation.

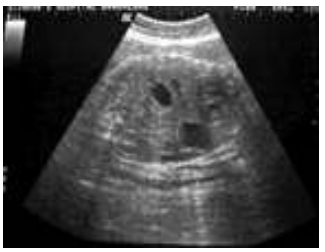
Dr. Hema Divakar, Dr. Chitra Ganesh, Dr. Ramesh. Divakars Hospital, Bangalore

Case - II :

Mrs.B, 27 years, primi gravida, non consanguineous and with a normal family history, had a mid trimester scan (18-19 weeks) which was reported normal. III trimester scan (33-34 weeks) had the following findings:

- Dilated fetal stomach and the first part of fetal duodenum.
- Dilated duodenum showed hyper peristalsis and a tapered distal n arrowing.
- Remaining bowel loops were normal.
- Liquor volume was normal.
- No other anomalies were detected.





Provisional Scan diagnosis:

- Dilated stomach and first part of duodenum with hyperperistalsis.
- In the absence of polyhydramnios the possibility of total duodenal obstruction was unlikely.
- The possibility of the subtotal obstruction due to band or annular pancreas was considered. The scan findings were suggestive of Incomplete duodenal obstruction due to malrotation / duodenal stenosis.

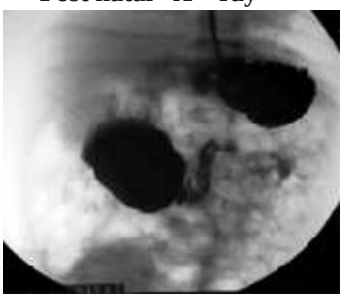
The parents were counseled regarding karyotyping to rule out trisomy and post natal follow – up of the baby. Patient underwent an emergency LSCS for PIH and delivered a female baby weighing 1.8kgs.

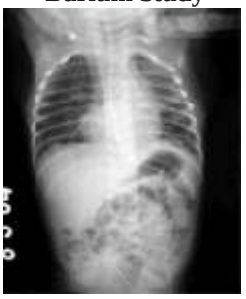
As per the advice of the Paediatric Surgeon, the baby was shifted to tertiary neonatal care center for further investigations and management.

Post natal Investigations:

- X-ray studies with the feeding tube inside, appeared normal. • Contrast Barium studies revealed distended stomach and grossly distended proximal duodenum with the dye passing through, was highly suggestive of a web.
- Fetal Karyotype was normal.

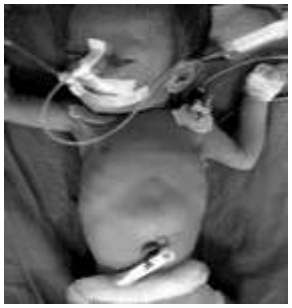
Post natal X – ray & Barium Study

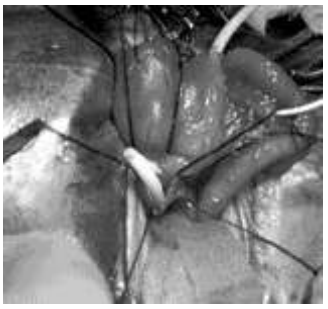




Final Diagnosis: Duodenal web.

Management:
The web was surgically removed and now the baby is doing well.





We wish all our members a very
Happy New Year 2005.
Together we shall strive to achieve
the goals set by the BDRI.