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Case Reports

Congenital Acinar Dysplasia (CAD): Case Report of an Extremely Rare Cause of Pulmonary Hypoplasia

ABU FOIZ MOHAMMAD MIZANURRAHMAN[1], AHMED AL ZOANI[2], AAHMED AL AMERI[3]

Introduction

The primary function of lung is to accomplish exchange of oxygen & carbon dioxide to accommodate the need of aerobic cellular respiration . To accomplish this a large thin alveolar capillary membrane is required.[1]Lung development takes place in 5(five) phases like 1. Embryonic phase 2.Pseudoglandular phase 3.Canalicular phase 4. Saccular phase and 5.Alveolar phase.[1]

CongenitalAcinar Dysplasia (CAD), an extremely rare form of pulmonary hypoplasia, is a lethal condition characterized by arrest of lung morphogenesis at the end of Pseudoglandular phase leading to non development of acinar structure distal to terminal bronchiole resulting in absence of gas exchange betweenlungs and its circulationTreatment of this condition is limited to supportive care.We are going to describe a case of lethal pulmonary hypoplasia caused by CAD.

Case Report

A 2.44 kg female baby at 36 weeks gestation was delivered by elective repeat lower segment cesarean section, the product of a consanguineous marriage.Mother wasa 30 yrs oldSaudi woman $G_4P_3A_0L_2$with a history significant for a previous neonatal demise despite aggressive respiratory support including surfactant replacement therapy.Maternal medical and labor history were unremarkable.The infant was non vigorous at birth with a nuchalcord noted, and required positive pressure ventilation.APGAR score was 6 & 8 at1 & 5 minutes respectively. Cord blood gas was P^H 7.27, PCO_2 41.6, BE -7, HCO_3 17.2.Initial physical examination demonstrated an appropriate gestational ageinfant with no distress, and was admitted to the well baby nursery.

At 3 hours of age, the infant was noted to be tachypneicand required oxygen prompting admission to NICU.Capillary blood gas from baby showed P^H 7.29, PCO_2 37.3, BE -7.6, HCO_3 17.7. CXR showed bilateral haziness.

The infant was intubated for progressive hypoxemic respiratory failure and responded to one dose of surfactant, leadingto extubation within 24 hours.The respiratory status deteriorated within 48 hours with CXR (Fig.1) demonstrating bilateral opacities leading to reintubationand mechanical ventilation with additional surfactant administration with partial and

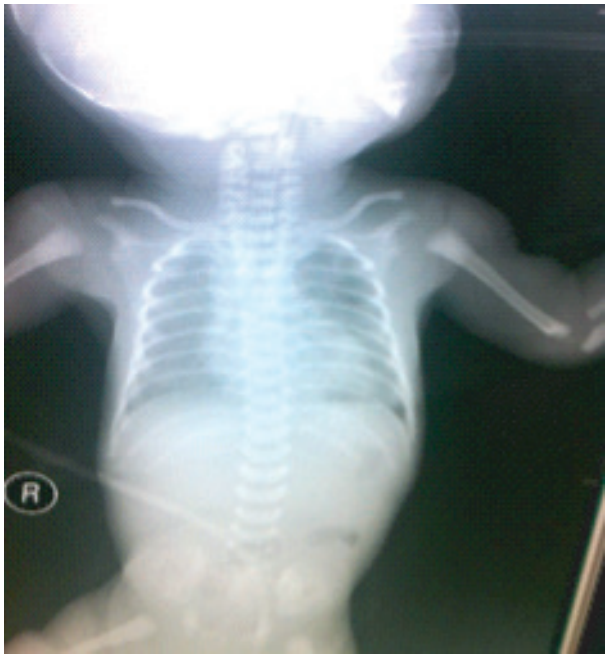

Fig.-1: *CXR of Congenital Acinar Dysplasia*

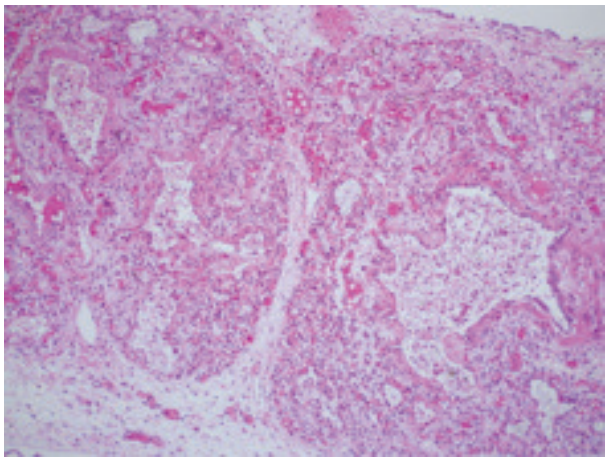

Fig.-2. *Histopathological picture of Congenital Acinar Dysplasia*

1. Specialist Pediatrics & Neonatology, NICU, Abha General Hospital, Abha, KSA.
2. Consultant Neonatology, NICU,Abha General Hospital, Abha, KSA.
3. Consultant & Head of NICU ,Abha General Hospital, Abha, KSA.

Correspondence: Dr. AFMM Rahman, Specialist Pediatrics & Neonatology, NICU, Abha Maternity and Children Hospital, Abha, KSA. E-mail : drmizan007@hotmail.com

transient responses to each dose.A sepsis work up was performed and broad spectrum antibiotics were initiated.Echocardiogram showed moderate size PDA with no evidence of PPHN.

She was subsequently switched to high frequency oscillatory ventilator on day of life 12 for worsening respiratory failure and required bilateral chest tube placement following the development of pneumomediastinum, pneumopericardium, pneumothorax,and subcutaneous emphysema over the neck and upper chest.

She died following an acute respiratory arrest on the following day unresponsive to resuscitation.

Bilateral lung biopsy which was taken after the death of the baby showed features consistent with CAD. (Fig. 2&3)

مستشفى الملك فيصل التخصصي ومركز الأبحاث
King Faisal Specialist Hospital and Research Center
KFSHRC_REF

Referral MRN: KFSH MRN:90228772
Requisition #: Accession #:SP-12-006669
Patient Name: Saeed, Eida Salem
Sex: Female Age: 3 months DOB: 25-May-12
Referring Client: KFSHRC Reference Lab
Client Address: P.O. Box 3354 MBC#10
Phone # :Tel: (966-1) 442-4230 City: Riyadh 11211
Results To:

Surgical Pathology Report

Case Number: SP-12-006669 Collected Date/Time: 09-Jul-12 12:00:00
Responsible Pathologist: Al-Dayel, Fouad Hassan Received Date/Time: 09-Jul-12 12:59:00

Patient History

Referral from Abha General Hospital-Assir Region, MRN 86721. A 4-day-old female baby delivered by CS, looks nearterm (" weeks gestation), observed to have tachypnea and distress three hours after delivery. Family history of one baby who died one year back after six days of life, full term with possibility of surfactant protein B deficiency.

Specimen Submitted

1. RIGHT LUNG BIOPSY
2. LEFT LUNG BIOPSY

Gross Description

1. The specimen is received in formalin labeled biopsy of right lung. It consists of two fragmented pieces of wedge like pale tan soft tissue measuring 1 x 1 x 0.3 cm and 1.3 x 1 x 0.3 cm. Bisected and entirely submitted in one cassette.

2. The specimen is received in formalin labeled biopsy of left lung. It consists of two fragmented pieces of pale tan wedge like soft tissue measuring 0.5 x 0.4 x 0.3 cm and 1.1 x 0.8 x 0.4 cm. Bisected and entirely submitted in one cassette.

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Diagnosis

IGHT LUNG BIOPSY:

MORPHOLOGY IS CONSISTENT WITH ACINAR DYSPLASIA.

2. LEFT LUNG BIOPSY:

MORPHOLOGY IS CONSISTENT WITH ACINAR DYSPLASIA.

Dr. Mizan
specialist NICU
Code No.154 & P. 157

Al-Dayel, Fouad Hassan, Consultant
(Electronically signed by)
Verified: 08-AUG-2012 15:17
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Fig.3:*Histopathology report of Congenital Acinar Dysplasia*

Discussion

Congenital AcinarDysplasia (CAD) isa lethal developmental disorder of the lung and a rare cause of primary pulmonary hypoplasia.Lung development begins early in human gestation (by day 25) and extends well into childhood. The primary goal of lung development is to create a large gas exchange area with a thin air-blood barrier. This is achieved by branching of the airways to form the conducting and proximal respiratory airways and by septation to subdivide the airspaces into alveoli. Development of lung takes place in 5 (five) stages,these are 1. Embryonic phase2.Pseudoglandular phase3. Canalicular phase4. Saccular phase and 5. Alveolar phase.[1,2]The timing of these stages is not always fixed and considerable overlap may occur between stages.[1]

Embryonic phase begins with the development of lung bud on the ventral surface of the foregut just caudal to the laryngotracheal sulcus at 25 days of gestation and extends upto 7 weeks of gestation. The lung bud continue to grow by dividing in a dichotomous fashion giving rise to the conducting airways and five primordial lung lobes (two on the left and three on the right). The pseudoglandular phase extends from 7 to 16 weeks of gestation, resulting in completion of all 16 division of bronchi responsible for gas conduction. The canalicular phase extending from 16 to 28 weeks is responsible for transformation of previable lung to the potentially viable lung that can exchange gas. Three major events takes place during this stage are – a. appearance ofacinus, b. epithelial differentiation with the development of potential air-blood barrier, andc. beginning ofsurfactant synthesis withintype II cells.[2]*Acinus* is the gas exchange unit of the lung and encompasses a respiratory bronchiole and all of its associated alveolar ducts and alveoli.A terminal bronchiole with all of its associated acinar structures constitutes a *lobule.*[1]

The saccular stage encompasses the period of lung development from28 weeks to term. The terminal sac or saccule is the distal airway structure that is elongating, branching and widening until alveolarization is completed, which is initiated from the terminal saccules by the appearance of septae in association with capillaries , elastin fibres, and collagen fibres.[2]

Alveoli begin to appear at about 32 weeks of gestation, most alveolar development occurs postterm.At the time of birth a term infant has 30% of adult number of alveoli. The lung grows postnatally mainly by an increase in alveolar number, and by 4 years of age the adult number of alveoli a total of between 200 million and 600 millionis formed. The subsequent increase of lung volume and surface area is due to increase in alveolar size.[1-3]

Factors which may delay or interfere with alveolarization are mechanical ventilation, antenatal and postnatal glucocorticoids, proinflamatory mediators hyperoxia or hypoxia and poor nutrition. WhereasVitamin A (retinoids) and thyroxine stimulate alveolarization.[2]

Pulmonary hypoplasia can result from primary defects in lung morphogenesis as well as from neurologic diseases associated with decreased fetal breathing movements, derangements of chest wall that restrict fetal breathing movements, renal disorders that compromise amniotic fluid volume and thereby restrict fetal breathing, or space occupying masses that restrict lung growth. Earlier the interference more severe is the pulmonary hypoplasia.[1]

In CAD lung development is arrested at the end of Pseudoglandular stage(16 week of gestation). As a result lung is developed upto the level of terminal bronchiole. No structure develops beyond this level. So baby will be delivered with hypoplastic lung with the absence of gas exchanging unit ‘acinus’, leading to ventilation failure and death.

Diagnosis of pulmonary hypoplasia can be made by (1) Lung to birth weight ratio of $\leq$0.9%, and (2) RAC (Radial alveoli count) $\leq$4.1 , which is defined as the number of alveoli cut by a line from the respiratory bronchiolar epithelium to the nearest connective tissue septum.[6]

The incidence of congenital pulmonary hypoplasia is 1 in 1000 live birth; this includes both primary and secondary pulmonary hypoplasia.[4] But the incidence of pulmonary hypoplasia due to CAD is unknown and seems to be under recognized, because of diagnostic difficulty. From 1986 to 2004 only seven cases have been reported with a definitive diagnosis of CAD. Of those, six were female, and the longest survivorlived for 2months.[5] Of noteour case is also a female baby. The etiology of CAD is not well understood. A genetic component seems likely, but a definite pattern is not apparent from the small number of documented cases.[5] From the cases reported so far it appears that females are more affected than males (9:1). So far 3(three) male babies were reported to have this

form of pulmonary hypoplasia, one of them delivered in Alhada hospital Taif, K S A.[8]It also appears that acinar dysplasiahas a tendency to recur in families in 40% of cases, suggesting an autosomal recessive pattern of inheritance. There is a case report of identical twins with acinar dysplasia.[7] CAD is diagnosed by exclusion of all other causes of pulmonary hypoplasia and a summation of clinical, imaging and histopathological findings such as in our case. Without autopsy this condition can be missed very easily because it can't be differentiated clinicallyfrom congenital alveolar dysplasia and alveolar capillary dysplasia.In both of these two conditions development of lung is arrested at the canalicular stage. In congenital alveolar dysplasia there is extreme retardation in alveolar development and in alveolar capillary dysplasia there is capillary misalignment and medial muscular thickening of the small pulmonary arterioles.[5] Congenital acinar dysplasia should also be differentiated from surfactant protein B deficiency (SP-B deficiency) and type-3 cystic adenomatoid malformation (CAM). Lamellar bodies are absent in infants with surfactant protein B deficiency. The presence of dysplastic lung tissue on histopathology and the presence of lamellar bodies in electron microscopy support the diagnosis of CAD and excludes the diagnosis of SP-B deficiency.[8] Congenital cystic adenomatoid malformation is due to an overgrowth of terminal bronchioles with cysts of various sizes and nodevelopment of normal alveoli.Cartilage is absent in type 3 CAM and it is a localized lesion and it compresses adjacent unaffected lung tissue while CAD causes generalized lung hypoplasia.[8,9]

Conclusion

In conclusion hypoxemic respiratory failure in a newborn baby represents a diagnostic and therapeutic challenge. Rare causes of pulmonary hypoplasia including CAD should be considered in the neonate with respiratory failure not responsive to maximum medical therapy. An autopsy is essential to the diagnosis of these causes of hypoxemic respiratory failure.

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Surgical Placement of Permanent Pacemaker Following Temporary Trasvenous Pacemaker in a Child with Congenital Heart Block and Moderate Size PDA: A Case Report

KAZI ZAHIDUL HOQUE[1], MASUMUL GANI CHOWDHURY[2], ABU SAYEED MUNSI[3], REZOANA RIMA[3], MANZOOR HUSSAIN[4]

Introduction

Congenital complete atrioventricular block (CCAVB), with incidence of 1/15000-20000 live born, is characterized by no transmission of any atrial electrical impulses to ventricles. It is almost always due (90% of cases) to the transfer of auto-antibodies (SSA/Ro and SSB/La) from SLE - or Sjogren's syndrome- affected mothers, even during their pre-clinical phase of the disease.[1-3] These auto-antibodies are responsible of ventricular endocardium damage and subsequent endomyocardialfibroelastosis. This can cause intrauterine miscarriage, with under-estimation of the real incidence of the disease or, fetal third-degree block, prolonged QT and Wolff Parkinson-White syndrome.[2]Third-degree block is frequently diagnosed during pregnancy, around 16-18 weeks of gestation. Timing of delivery as well as type and time of pacemaker implantation after birth are still controversial issues. Pacemaker implantation is indeed the only treatment for third-degree block and it is immediately required in presence of prenatal hydrops, low ventricular rate (<45/bpm) with no response to inotropes, and/or ventricular dysfunction.[3] In very low birth weight infants, because of the size of generators, a staged pacing strategy has been used, with temporary epicardial pacing wires, followed by definitive implantation when neonatal body weight reaches 2000g. Some cases have also been treated with permanent pacemaker implantation.[3-5]

1. Assistant Professor, Pediatric Cardiac Surgery, Dhaka Shishu (Children) Hospital
2. Assistant Professor, Pediatric Cardiac Surgery, Dhaka Shishu (Children) Hospital
3. Assistant Professor, Pediatric Cardiology & Interventional cardiology, Dhaka Shishu (Children) Hospital
4. Assistant Professor, Pediatric Cardiology & Interventional cardiology, Dhaka Shishu (Children) Hospital
5. Director and Professor and Departmental Head, Pediatric Medicine & Pediatric Cardiology, Dhaka Shishu (Children) Hospital

Correspondence: Dr. KaziZahidulHoque, Assistant Professor, Pediatric Cardiac Surgery, Dhaka Shishu (Children) Hospital, Sher-e-Bangla Nagar, Dhaka, E-mail: kzhoque72@yahoo.com

Case Report

A 9-months-old female child, born with the history of parents consanguineous marriageby caesarian section in a hospital with a birth wei-ght of 3.3kg, presented with bradycardia (heart rate of 30-35 beats/ min) and respiratory distress and with multiple syncopal attacks since her 8 months of age, for which she was admitted to Paediatric Cardiology unit of Dhaka Shishu (Children) Hospital. His mother, Gravida 2 and Para2, had no past medical history significant for Systemic Lu-pus Erythematosusor Sjogren syndrome. Her previous baby was in good health and no major congenital anomaly present. She has reported that the baby was prenatally diagnosed bradycardic by ultrasonogram and they were informed about the condition of the fetus.The parents also reported a history of repeated syncopal attacks and respiratory tract infections since the age of one month. Prior to that the child under treatment with diuretics and captopril and antibiotics.Clinical examination showed a continuous machinery murmur 4/6 at the left 2nd intercostal space. Femoral pulses weresymmetrically palpable. The infant was adequately nourished. Her psychomotor development was normal according to her chronological age. The first ECG showed (Fig.-2)3rd degree heart block with complete arioventricular dissociation.The child was stabilized in the cardiology unit and kept on two liters of oxygen through nasal cannula for respiratory distress. She was tolerating feeds. Her heart rate ranged between 30-35 beats/ min despite being given two doses of atropine (0.1 mg/dose). Chest X-ray AP view showed increased diameter of the cardiac shadow(Fig.-1**)**. Her electrocardiogram (ECG) showed features of complete heart block **(**Fig.-2), with the features of complete atrioventricular dissociation and heart rate was 34/min. Based on her condition, it was decided to do further evaluation and management (including the possi-bility of pacemaker insertion) to support the patient in the best possible way.

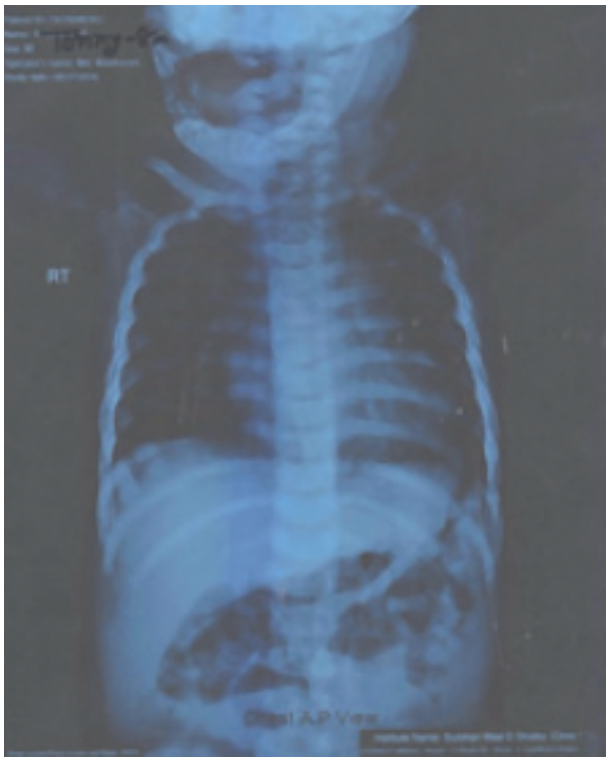

Fig.-1: *Preoperative X Ray Shows increased Cardiac Shadow and Plethoric Lung field*

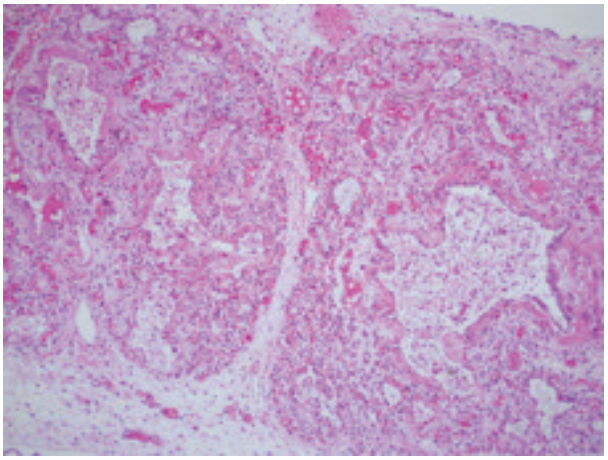

Fig.-2: *ECG before placing any pacemaker shows 3rd degree heart block*

Echocardiography was performed and it revealed a PFO with moderate size PDA shunting left to right. Rate showed extreme bradycardiac, no other abnormality detected.Considering the emergency situation intervention cardiology unit decided to put a temporary transvenous pacemaker. They ultimately implanted it on the 4th day of admission through the right femoral vein route and placed it into the right ventricular apex under general anesthesia, and the other end of the lead was connected to an external pacemaker generator and set the rate at 90 b/min with output 5 mA and capture was lost in 1 mA.The patient had developed repeated ventricular tachycardia (VT) during the procedure but no neurological abnormality detected after the procedure. We the cardiac surgery team decided to do a left lateral thoracotomy. Through that incision we ligated the PDA and implanted a permanent VVI pace maker at the apex of the heart. The decision of lateral thoracotomy was made on the basis of convenience for the PDA ligation otherwise it could be done through median sternotomy.At 7th admission day upon the availability of permanent pacemaker, baby was taken to operation theater, a left lateral thoracotomy was made at 4thintercostals space. A moderate size PDA was found after dissection of the fascia over the aorta. Multiple ligatureswere made to close the PDA (Fig.-5 &6). Pericardium was incised with a caution forpericardiacophrenic nerve and vessels. A permanent pacemaker (St. Jude VVIR PM with auto-capture at rate 90 beats/min) implantation was done and set the rate at 90 b/minutes and the generator was placed in the abdomen(Fig.-7 & 8) after making a subcutaneous pocket. Chest X ray (Fig.-4) shows the placement of lead and generator. The temporary pacemaker was disconnected as soon as commencement of the permanent pacemaker. A 12 lead ECG was done (Fig.-9) in the postoperative recovery unit.The ECG shows heart rate 90b/min with frequent pacemaker capture beats. Upon further follow up, the patient was stable and his general health was good. He was thriving well with normal developmentin subsequentfollow-up. The echocardiographic examination showed good left ventricular contractility with normal internal dimensions. There was no residual flow through the closed PDA.

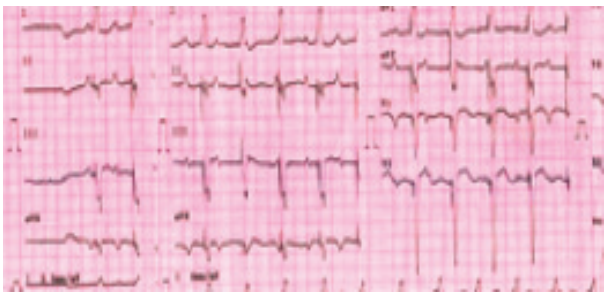

Fig.-3: *ECG after placing temporary pace maker. Heart rate shows 90b.min and Pacemaker capture is visible*

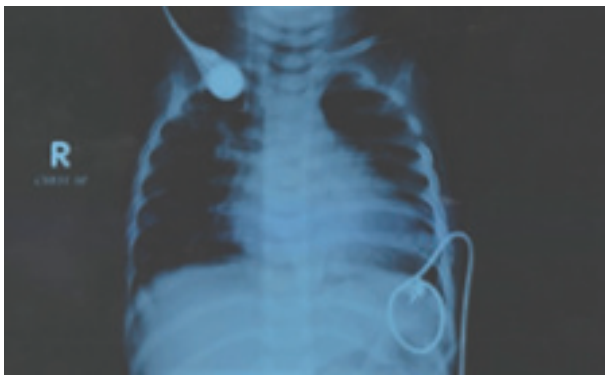

Fig.-4: *Chest X ray after surgical implantation of permanent pacemaker*

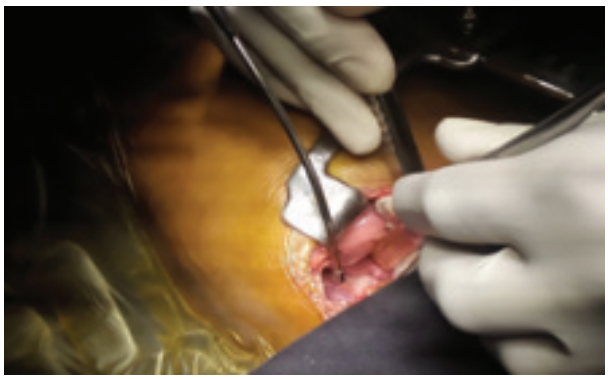

Fig.-5: *Lateral thoracotomy for PDA ligation & pacemaker implantation*

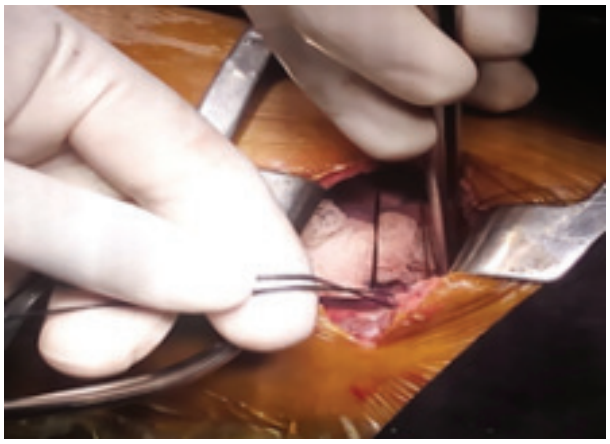

Fig.-6: *Multiple ligation of PDA*

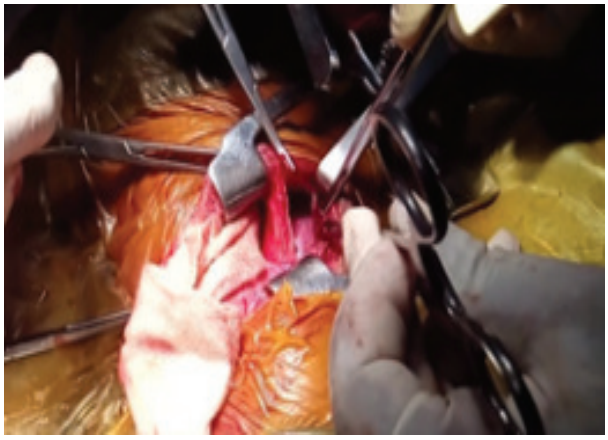

Fig.-7. *Incision through pericardium*

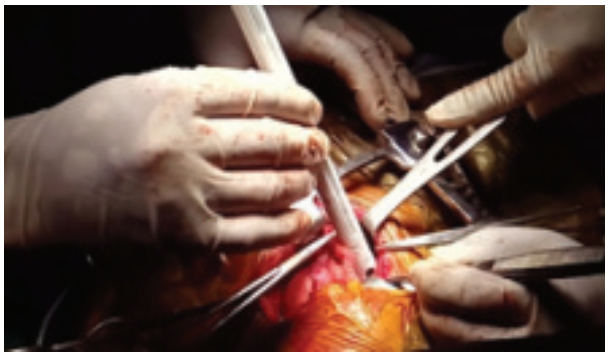

Fig.-8. *Pacemaker Implantation through lataral thoracotomy*

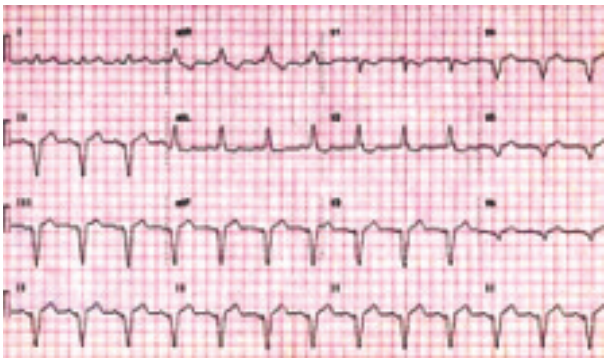

Fig.-9: *ECG after permanent pacemaker implantation. Hear rate is 90b/min and pacemaker capture is visible*

Discussion

It is well known that in both infants and older children permanent pacemaker leads may be implantedeither epicardially or transvenously. In small infants, when permanent pacemaker implantation is necessary epicardial leads are used. The reason for preferring the epicardial route is the patient's small body size. Transvenous lead implantation is hampered by anatomical peculiarities, which are often seen and include anomalous connection of the *venae cavae*as well as other complex endocardial anatomical lesions.[6] Apart from the procedural difficulties, such anomalies

also entail a risk of systemic embolism due to endocardial defects.[7] In addition, thrombosis in the superior *vena cava* is a common complication of endocardial lead placement.[8]

On the other hand, epicardial lead placement is more invasive. It involves subxiphoid section and possibly a partial sternotomy or thoracotomy. It is often complicated by post-pericardiotomy syndrome.[9] The usual epicardial leads are associated with a high incidence of rapidly increasing sensing and pacing thresholds after lead placement, necessitating the early replacement of lead and generator. Endocardial leads are favoured inolder infants with a body weight >8 kg,[10] or preferably15-20 kg,[11]. Moreover, insmall infants the small dimensions of the atrium[11] areinsufficient for successful placement of the preformedatrial lead.The elevated pacing thresholds and the high incidenceof exit block associated with conventional epicardialleads are caused by a combination of epicardial fibrosiswith scar formation, and/or pericardial adhesionsfollowing the surgical procedure. Cases havebeen reported of exit block due to lead fracture caused by the infant's muscular activity.[12]The five-year survival of the conventional epicardial lead is 40-70%.[13,14] In neonates and infants with a permanent pacemakerthe occurrence of episodes of loss of consciousnessmay be due to pacemaker malfunction.[15]Follow-upchecks should be performed every 6 months inthose without symptoms; parameters of pacemakerfunction should be measured, mainly the resistanceand threshold of the atrial and ventricular leads. Ifsymptoms occur as a result of pacemaker malfunction,24-hour Holter monitoring is useful for their detection.[16]Pacemaker malfunction due to the developmentof fibrosis around one or both of the epicardial leadscan be treated by the substitution of endocardial leads.At the same time, the generator can be left in its abdominalsite and the electrodes can be connected viaa subcutaneous channel.

Conclusion

In conclusion, the choice of lead type during pacemaker implantation should aim at achieving optimum cardiac function and maximum battery and lead life, while taking account of the risks of lead placement as well as the future surgical treatment of the patient.

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