Certificate

I hereby certify that I have performed/assisted all the procedures listed in the work record.

Simran Kundan.

SCTIMST, Thiruvananthapuram

Date: 02/10/2013

Forwarded: The above candidate has satisfactorily carried out the minimum required procedures.

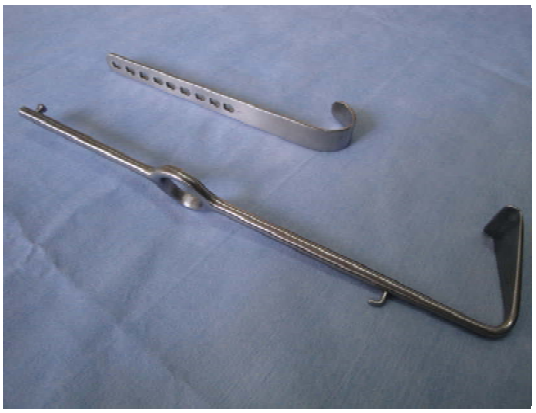
Jayakumar K. Seal.

Prof. and Head,

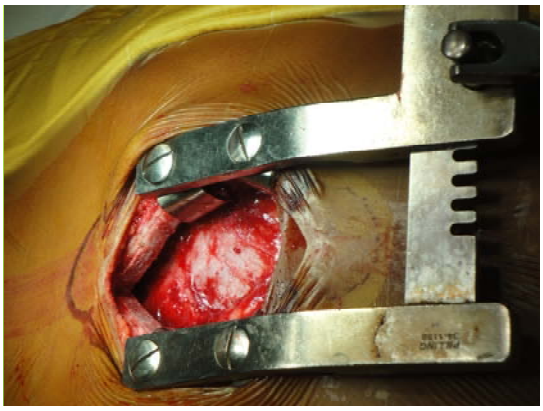
Department of CVTS,

SCTIMST, Thirvanathapuram.

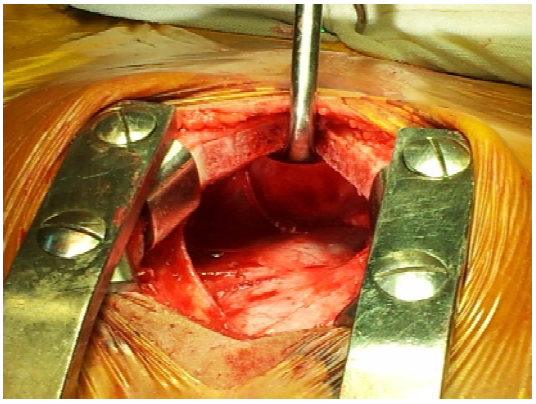
Tanned Pericardial Patch closure of OSASD via ministernotomy



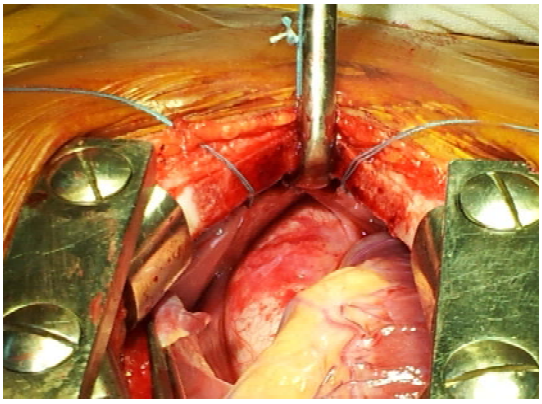
Sternal retractor designed at SCTIMST



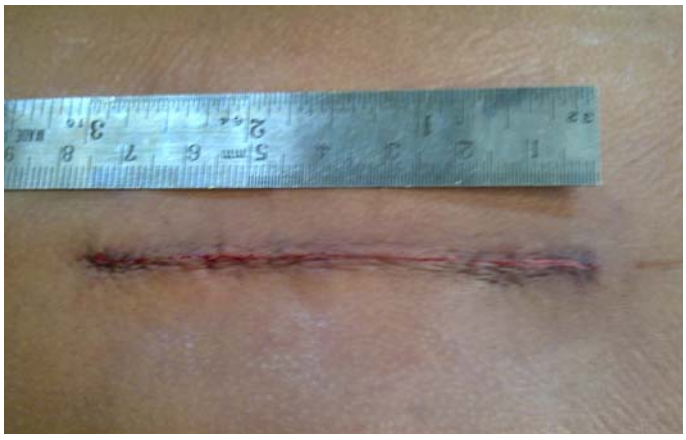
Lower sternotomy



Retractor lifts the sternum up



After placement of the central pericardial stay, aorta comes into view for cannulation



7.5 cm incision in a 16 Kg child.

Interesting Cases

1) Never loose Hope: Arterial Switch Operation with LIMA to LAD graft.
2) Synchronous Bentall Debono procedure with Hybrid TEVAR for dual Aortic pathology.
3) Foregin body in Ascending Aorta.
4) Complex congenital heart disease complicated by haitus hernia, a management dilemma.
5) Trans Esophageal Echocardiography with 3-D Reconstruction of Dehiscence of Starr-Edward valve Prosthesis in Mitral Position.
6) Iatrogenic LMCA Dissection.
7) Alfieri's Edge to Edge repair in a case of double inlet left ventricle (DILV) with moderate Atrio-ventricular valve regurgitation.
8) Differential saturations in 4 limbs, an unusual presentation.
9) Modified BT shunt post iatrogenic injury following attempted PDA stenting.
10) A Night mare session.

Never loose Hope: Arterial Switch Operation with LIMA to LAD graft.

One month old infant, diagnosed as d-TGA, multiple muscular VSD's, ASD regressed LV, normal coronaries from facing sinus, juxtacommissural LAD, was planned and taken up for ASO.

Standard Aorto-bicaval cannulation with blood cardioplegia, with core cooling to 22 deg, with a CPB time of 486 minutes and a Aortic cross clamp time of 258 minutes.

After dissecting the Aorta and PA's, PDA was interrupted, ASD closed with bovine pericardium and the VSD's closed directly.

Aorta transected and coronary buttons harvested, adequate length of epicardial coronaries mobilised. LAD origin was justacommisural and got dissected while proximal probing, after much deliberation we decided to continue with the procedure and addresses the issue after completing the rest of the procedure. The neo pulmonary artery was reconstructed with tanned pericardium and posterior commissure resuspended. Lecompte manoeuvre done.

Coronary buttons anastomosed at the intented site on the Neo Aorta, conal branch sacrificed as it was causing tension on the suture line. The neo Aorta thus constructed was anastomosed to distal Aorta.

Heart picked up in sinus bradycardia, hence paced ventricularly at 130/min. The child failed multiple attempts of weaning from bypass, finally managed to do so with milrinone 0.5, adrenaline and nor-adrenaline 0.1 mic/kg/min. LV contractility was extremely poor. Hence it was decided to revascularise the LAD.

Hence LIMA anastomosed to LAD using 8-0 prolene, Post grafting contractility improved marginally and the child was able to wean of bypass and shifted to ICU with milrinone, adrenaline, nor-adrenaline with the sternum stented.

Post procedure the child's recovery remained surprisingly uneventful. Expectedly post procedure the child had ST elevation with narrow complex QRS, which settled by post op day two. PD was inserted on the night of the surgery and on day one the membrane was opened for collection. Post op day 4 the child was taken for delayed sternal closure, which the child tolerated well, per-op LV contractility was visually good and apex was contracting well. The child was extubated on POD 9 and was maintained on non invasive ventilation for 4 more days and was shifted to ward on post op day 17.

Prior to discharge the the child had no regional wall motion abnormality and trivial AR, though ECG showed showed Q waves is $V_{4\text{-}6}$.

Discussion:

Bypass grafting is a rare procedure carried out in pediatric age group and few cases have been report in literature. Most were in similar life saving and called for drastic step situation as ours[1]. The largest series of pediatric coronary artery bypass comes from Mavroudis group, they have report 16 children undergoing ITA to coronary artery bypass[2], the reasons they cite for IMA to coronary artery grafting are post ASO, Kawasaki disease, congenital lesion, and iatrogenic lesion leading to poor LV function. Post ASO and iatrogenic injuries put together are the most common reason for this rare procedure[2]. All the patients they performed IMA to coronary artery grafting were post ASO and had stenosis of the LMCA. Brackenbury group reported 2 cases, which required LIMA to coronary artery bypass post ASO and they have cited technical difficulties as to implant the coronary buttons. Iatrogenic causes have been purported as the commonest cause of myocardial ischemia[4], proximal narrowing, and residual tension on the coronaries or dissection and ostial distortion.

The choice of arterial conduit is limited in this miniature subset of patient and the options of conduits include subclavian artery[5], gartro-epiploic artery. The results of use of these conduits is not known as there are only case reports, though they are a well know viable option in ALCAPA, there use in other situations is only desperate[1].

What this case taught us is never to loose hope and what ever the situation never give up, this child being discharged home is a living proof of that.

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3) Internal mammary artery to coronary artery bypass in paediatric cardiac surgery, Edward Brackenbury, Helena Gardiner, Kak Chan, Mark Hickey, European Journal of Cardio-thoracic Surgery 14 (1998) 639–642
4) Myocardial ischemia following arterial switch operation: An uncommon etiology. R Saileela, Shanti C, Krishna Manohar, Raghavan Subramanyan, and KM Cherian, J Thorac Cardiovasc Surg 1996;111:181-189.
5) Long-Term Results of Repair of Anomalous Origin of the Left Coronary Artery from the Pulmonary Artery. Rüdiger Lange, Manfred Vogt, Jürgen Hörer, Julie Cleuziou, Andrea Menzel, Klaus Holper, John Hess and Christian Schreiber. Ann Thorac Surg 2007;83:1463-1471.

Image 1: Donut of LAD which got avulsed on probing the LAD.

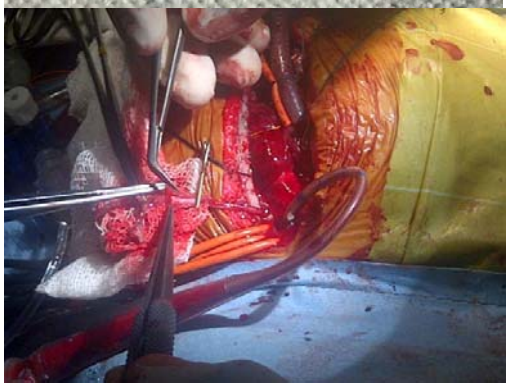

Image 2: Preparation of LIMA for implantation onto the LAD.

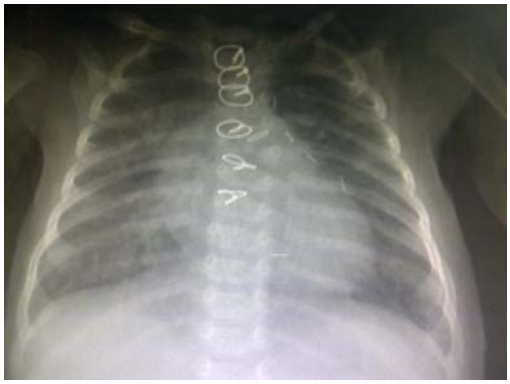

Image 3: Antero-posterior Chest x-ray of patient prior to discharge showing the unmistakeable trace of the clips along the LIMA to LAD anastomosis.

Synchronous Bentall Debono procedure with Hybrid TEVAR for dual Aortic pathology.

Introduction: Patients of Marfan's syndrome present with pathologies affecting the Aorta in the form of aneurysmal dilatation of the root with or without valve incompetency and or dissection. It is rare to encounter a patient with dual pathologies, aneurysm at one and dissection in the other domain of thoracic aorta that dictates personalised therapeutic paradigm. Herein we describe one such patient with severe aortic regurgitation and Aortic root aneurysm with Stanford Type B dissection managed using concomitant traditional open surgical repair with hybrid endovascular therapeutic strategy.

C**ase Report:**

A 38 year old female patient having known Marfan's syndrome, presented to us with dyspnoea on exertion NYHA class II, since 5 years but denied history of any episode of severe chest pain. On evaluation she was diagnosed to have ascending aortic aneurysm with aortic regurgitation and Stanford Type B aortic dissection.

2D ECHO showed dilated left ventricle dimension of 64/44 mm, septal dimension of 8/14 mm, posterior wall dimension of 8/30 mm, ejection fraction of 68%, severe aortic regurgitation, no regional wall motion abnormality. Computed tomography showed dilated aortic root measuring 5.6 cm, sino tubular junction measuring 5.3 cm, proximal ascending aorta measuring 4.0 cm with classic Stanford Type B aortic dissection with compressed true lumen and large false lumen totally measuring 6.6 cm in size. Intimal tear and intimo-medial dissecting flap starting immediately distal to the left subclavian artery and extending up to the aortic bifurcation. (Fig 1a and 1b)

The patient underwent Bentall's deBono procedure and hybrid TEVAR for these dual pathologies.

After exposing left common carotid and right common femoral artery, median sternotomy was performed and external cardiac findings noted were confirmed by Transesophgeal ECHO. High Aorto-right atrium bypass instituted, left ventricle vented via right superior pulmonary vein. After cross-clamping the aorta, aortotomy done and heart arrested using direct ostial blood cardioplegia.

Coronary buttons were harvested and prepared during core cooling. Composite graft was prepared using 26 mm collagen coated knitted polyester graft and 25 mm aortic Chitra heart valve prosthesis (TTK-Chitra®, TTK product, Chennai, India). Composite graft was then sutured to the aortic annulus using braided polyester sutures using interrupted technique. Coronary buttons were anastomosed to the neo ascending aorta and distal end of the neo-aorta was sutured to the native aorta using 4-0 polypropylene in a continuous fashion (Fig 2a). Left carotid artery was debranched using 10 mm polyester woven graft from ascending aortic graft to left common carotid in the neck and interrupting it proximally.

Total CPB time of 170 minutes, aortic cross clamp time of 103 min, with moderate hypothermia maintained at 27^0C.

Right common femoral arteriotomy was done and angiography performed showed patent open repair status. A 36 X 167 mm Talent (Medtronic AVE ®, Santa Rosa, CA, USA) aortic stentgraft was deployed just beyond innominate artery ostium to mid thoracic aorta (D8 vertebra). Post deployment angiography showed patent open repair status completely covered intimal tear, opened up true lumen and only faint filling of false lumen is abdominal aorta. (Fig 2b)

Patient recovered well from the procedure and was discharged from the hospital on 7^{th} postoperative day. On follow up, she is symptom free and CT angiogram shows patent open repair status with completely thrombosed false lumen. (Fig 3)

Discussion:

Marfan syndrome has dogged the aortic surgical community as to the optimal mode and timing of repair; the consensus for it is far from reached. Patients of Marfan's present with dilatation of the thoracic aorta predominantly with or without dissection[1] that need to be addressed as and when they so appear. Many investigators have reported this phenomenon so as to the mode and optimal timing of intervention/surgery for varied presentation and the long term follow up is necessary to decide appropriate further therapies[2].

Historically these patients were treated with staged procedures, usually treating the one presenting as emergency and addressing the quiescent pathology later[3]. With the advent of TEVAR a paradigm shift has occurred in the setting of Stanford B aortic dissection[2, 4]

Aortic root replacement is an established form of treatment for ascending aortic aneurysm with severe aortic regurgitation ever since described by Bentall and deBono[1, 5] in 1968 along with its various modifications described subsequently. Hitherto Bentall deBono for aortic root aneurysm with aortic regurgitation used to be performed along with elephant trunk (off late frozen elephant trunk) for a patient with similar clinical setting of our patient[6]. These procedures are complex with increased morbidity and mortality in view of need for hypothermic circulatory arrest and difficulty in manoeuvring prosthetic graft through the compressed true lumen of the dissected aorta, mandating an adjuvant procedure later[7]. TEVAR is fast evolving as a first line therapeutic strategy for type B aortic dissection across the globe. Therefore we believe open conventional procedure for aortic root coupled with synchronous hybrid TEVAR for type B aortic dissection has been a personalized combination with excellent immediate safety, nevertheless efficacy to be proved in due course on follow up.

To our knowledge this is the first reported case of aortic root aneurysm along with Stanford type B aortic dissection presenting together which has been successfully repaired with synchronous Bentall deBono procedure for aortic root aneurysm with Hybrid distal aortic arch repair for Stanford type B aortic dissection.

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2) Botta L, Russo V, La Palombara C, Rosati M, Di Bartolomeo R, Fattori R. Stent graft repair of descending aortic dissection in patients with Marfan syndrome: An effective alternative to open reoperation? J Thorac Cardiovasc Surg. 2009 Nov;138(5):1108-14.
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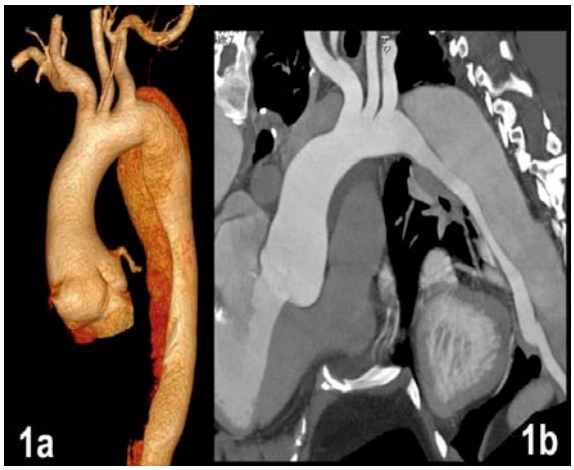

Figure 1a: CT angiogram 3D volume rendered image, showing dilated aortic root and proximal ascending aorta along with Stanford Type B aortic dissection.

Figure 1b: CT angiogram, multiplaner reconstruction showing dilated aortic root and proximal ascending aorta with Stanford Type B aortic dissection with severely compressed true lumen.

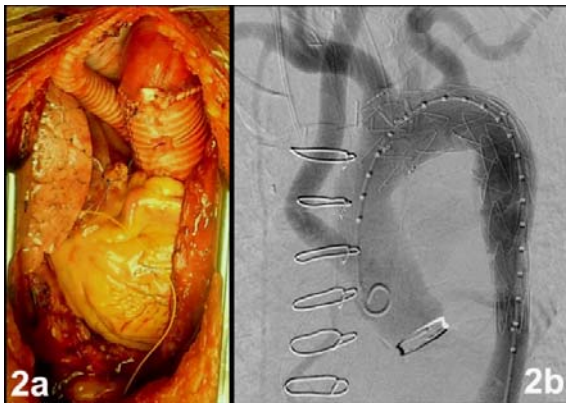

Figure 2a: Per operative picture showing a 26 mm polyester ascending aortic graft with a 10 mm side limb to left common carotid artery.

Figure 2b: Completion angiography following TEVAR showing patent ascending aortic graft with prosthetic aortic valve and good flow to coronaries and left common carotid graft with interrupted proximal left common carotid and a 36 X 167 mm aortic stent graft in situ.

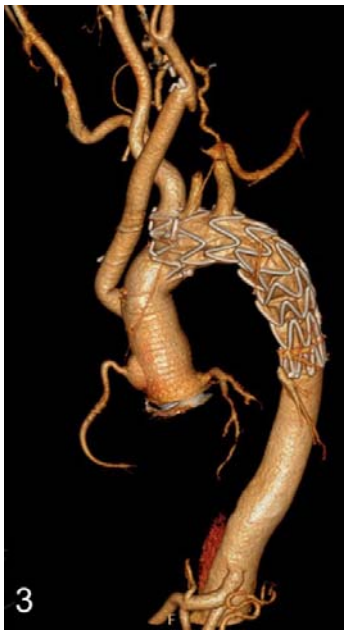

Figure 3: Follow up CT angiogram 3D volume rendered image, showing ascending aortic graft, and aortic stent graft in flush with innominate artery with completely obliterated false lumen and opened up true lumen.

Foregin body in Ascending Aorta

A 22 yr old male who was involved in a road traffic accident 7 months prior to the visit to our institute, he sustained a fracture of the right clavicle, for which he underwent K-wiring, after a period of 2 months the pins were scheduled for removal. However at manipulation one of the pins got further embedded in the bone and he was rescheduled for removal under anesthesia.

In the mean time he developed dysopnea on exertion functional class II, with pedal edema, chest x-ray revealed the pin had migrated into his chest, along with Right pleural effusion. Lateral xray was more informative so as to reveal the location within the cardiac silhouette.

Echo showed a hyperechoic shadow in the ascending Aorta which was tenting the RCC and causing severe AR. Flouroscopy revealed the pin to be moving with each heart beat. The patient was taken up for exploration.

Per-op the patient surprisingly had no pericardial effusion. Externally the point of entry was at the right sterno-clavicular joint, internally the nail had penetrated the pericardium above the SVC and the Ascending Aorta on it anterior surface forming a saccular aneurysm. TEE showed the nail to be tenting the RCC and causing severe AR. The nail was removed, TEE showed the AR to be persistent and RCC damaged. Hence it was decided to go on pump and examine the Aortic valve.
On examination, the RCC was found to been torn and not feasible for repair. Hence it was decided to replace the aortic valve with 19 mm Chitra TTK® heart valve prosthesis.

Three months post op the patients is doing well and is in functional class I.

Discussion:

It is extremely rare to see a patient with a foreign body penetrate the heart or great vessels without causing tamponade[1]. The management of foreign bodies embedded in-vivo is based on the size of the foreign body. Medical management should be pursued if the patients is asymptomatic, foreign body is small and smooth and risk of contamination is minimal[2]. Smaller the foreign body (< 3mm) impacting the heart greater the risk of embolisation and earlier should the patient be subjected to surgical extraction to prevent embolization[1]. In management of these patients it is imperative to localize the foreign body[3] prior to embarking on a virtually impossible and equally frustrating surgical expedition of the vascular system because foreign bodies which get lodged in the vascular system are notoriously known to embolize [3, 4]. In our patient the foreign body could be easily taken out by taking a purse string around the site of entry into the Aorta to control it, we needed to go on bypass only to replace the aortic valve.

The TEE images guided the surgery throughout its course, it perfectly delineated the site of impaction and the mechanism of AR[5].

References:

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constrictive pericarditis. Ann Thorac Surg 1998;65:1786–1787.
3) Foreign bodies in the heart: surgical or medical therapy? Guglielmo M. Actis Dato, Enrico Aidala and Giuseppe F. Zattera, *Ann Thorac Surg* 1999;68:291-292 (Letter to Editor)
4) Survival After Embolization of a Complete Prosthetic Aortic Valve to the Aortic Arch, John M. Stoneburner, Jr, MD, Bernard L. Tucker, MD and Richard J. Hurvitz, MD. Ann Thorac Surg 1990;49:140-2.
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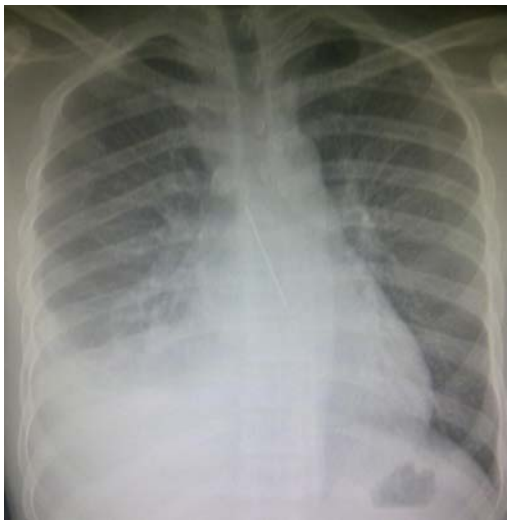

Figure 1: AP view showing the foreign and right pleural effusion.

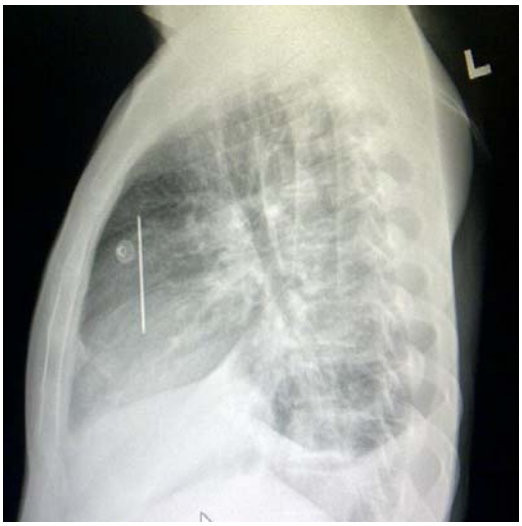

Figure 2: Lateral chest xray showing the foreign body within the cardiac silhouette.

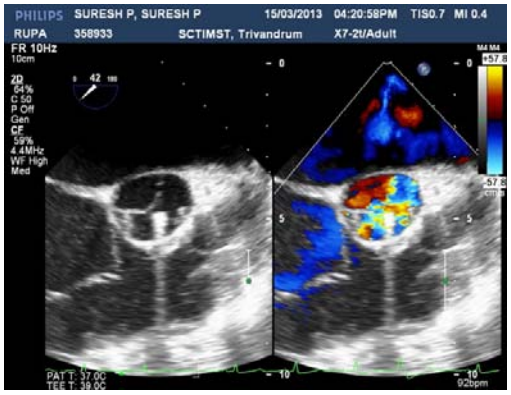

Figure 3: Intraop TEE showing the foreign Body tenting the RCC and causing sev AR

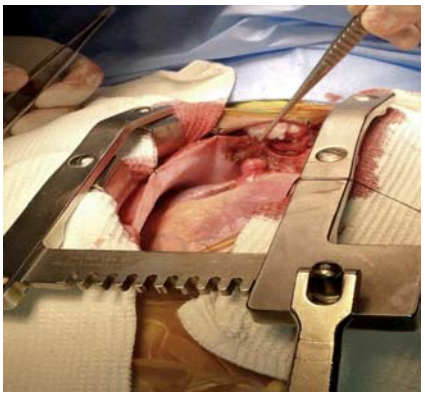

Figure 4: Intraop picture showing the point of entry just above the SVC and causing a pseudo aneurysm at its point of entry into the aorta.

Figure 5: The extruded K wire.

Complex congenital heart disease complicated by haitus hernia, a management dilemma.

Case:

We present the case of 18 month child weighing 5.7 kg, who presented with heterotaxy syndrome, asplenia complex, supracardiac total anomalous pulmonary venous (TAPVC), complete unbalanced atrio-ventricular canal defect (AVCD) amounting to single ventricle, d-Transposition of Great Arteries (d-TGA), with pulmonary atresia (PA), RPA stenosis.

On pre-operative evaluation the chest X-ray showed a left sided retrocardiac shadow. On further imaging it showed a rolling type of hiatus hernia.

Child was having severe cyanosis and was taken up for TAPVC rerouting, BDG with atrial septectomy, we did not address the Hernia as it was sliding in nature and aymptomatic[1]. (Picture 1, 2)

The patient failed extubation twice, having bronchospasm, initially we thought in terms of a failing Glenn as the reason for the respiratory failure, a cardiac catheterisation and Aortogram proved the Glenn pressures at 16 mmHg and insignificant APCA's to both the lungs. We then profiled the Hernia on fluoroscopy after injecting contrast medium via the naso-gastric tube. The herniated stomach showed up unmistakably with its ruggae. (Picture 3).

Discussion:

Heterotaxy syndrome and TAPVC is associated with high early and late mortality[2], hiatus hernia has been associated with heterotaxy syndrome[1] and the mortality is no different with or without the hernia[3].

Management of haitus hernia depends on whether the hernia is symptomatic or rolling in nature. Timing of hernia repair is debatable[3] especially in heterotaxy syndrome. In our patient the hernia was indeed causing symptoms of a space occupying lesion and needed to be addressed[3]. The reason the hernia was not addressed at the index surgery because we thought that post heparinization to do an extensive dissection aound the hernia sac would not be prudent, and technically to do the surgery via the sternotomy the hernia sac and the cardiac mass have to be on the opposite side of each other.

In the post op period the hernia may become overt, presenting with resistant respiratory distress as a result of compression of an already compromised lung[4]. Another mechanism of respiratory distress, especially pertinent to our case would be the compression of the left atrium[5] leading to an increase in LVEDP. During positive pressure the hernia reduced but after extubation the patient's lungs got progressively compressed.

A hernia causing compressive symptoms needs to be addressed and the strategy should probably be surgical[6].

References:

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5) Konstantinos C Koskinas, Kostas Oikonomou, Eleni Karapatsoudi. Echocardiographic manifestation of hiatus hernia simulating a left atrial mass: Case report
Cardiovascular Ultrasound 2008, 6:46
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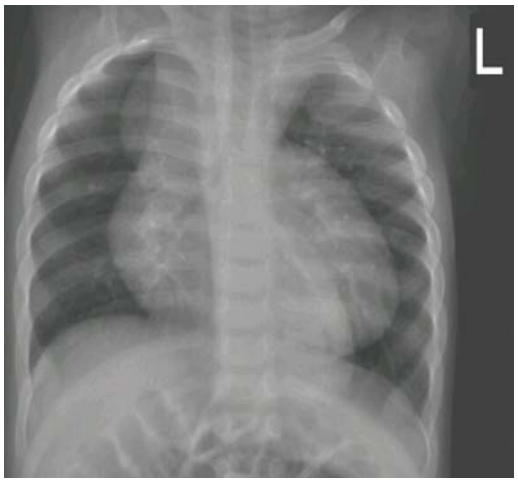
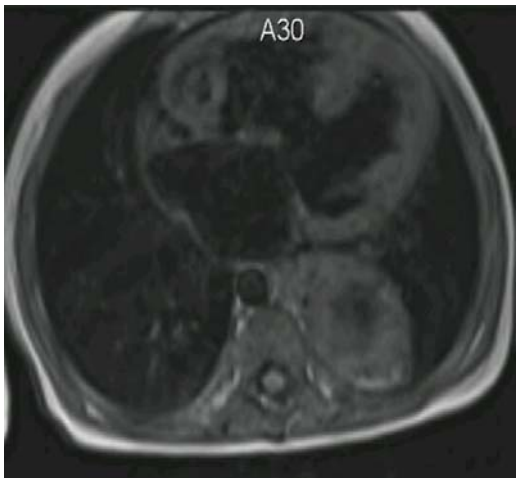
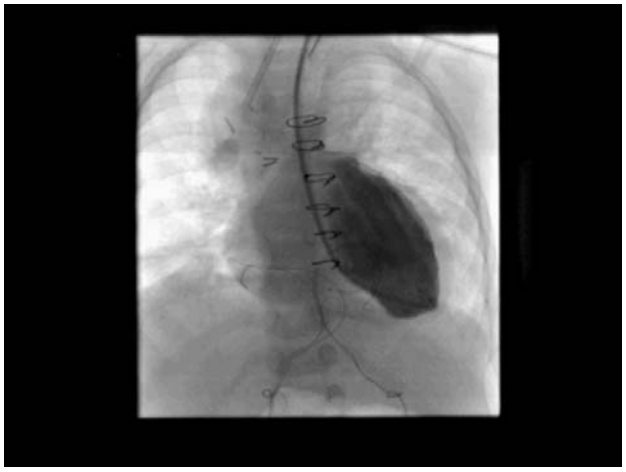


Picture 1: Chest X-ray PA view-showing the Hernia masquerading as left lower lobe collapse.



Picture 2 a and b: MRI image showing retrocardiac gastric bubble.



Picture 3: Catheterisation study with dye injected via feeding tube-showing the retro cardiac stomach.

Trans Esophageal Echocardiography with 3-D Reconstruction of Dehiscence of Starr-Edward valve Prosthesis in Mitral Position

A 55 year old male who underwent double valve replacement in 2005, following which he developed pseudomonas aeruginosa infective endocarditis and underwent a re-double valve replacement with in a period of 45 days. He was asymptomatic for 5 years. The patient was re-admitted with features of pulmonary edema. He was afebrile and WBC count and ESR were normal. Trans Thoracic Echocardiogram showed severe para-valvar mitral regurgitation (MR) and fluoroscopy showed showed rocking movements of the Starr Edward (SEP) valve. Patient was taken up for a re-operative mitral valve replacement. Intra operatively the Trans Esophageal Echocardiography (TEE) showed the annulus was almost completely dehisced, with rocking of the SEP and the valve was held in position by a few sutures with severe para-valvar MR. Pre-operative Blood cultures and intra-operative tissue cultures did not grow any organism. Patient made an uneventful recovery.

Discussion:

Foster et al described a technique to identify the location and extent of para-valvar MR by superimposing the face of a clock on the valve. The aortic valve is defined as the 12 o'clock position and the LA appendage lies in the 10 o'clock position. We had used this technique and imaged the prosthetic valve with the 3 standard mid-esophageal ventricular views

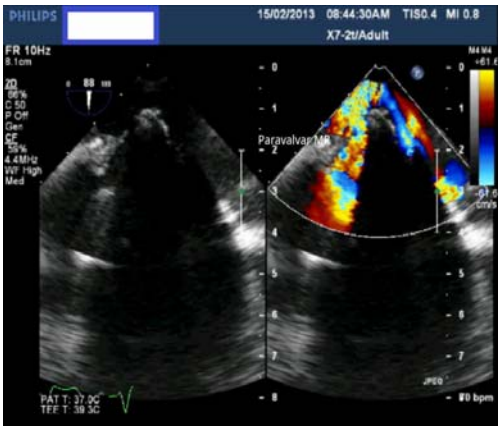
1) Four-chamber view showing rocking motion in both 2 & 8 o'clock)
2) Two-chamber view showing dehiscence at 4 o 'clock) and
3) Long-axis view demonstrating significant para-valvar space at 6 o'clock).

From the above 3 mentioned mid-esophageal views it was evident that the prosthetic valve had a dehiscence of approximately 50% of its circumference with attachment at the level of aortic root.

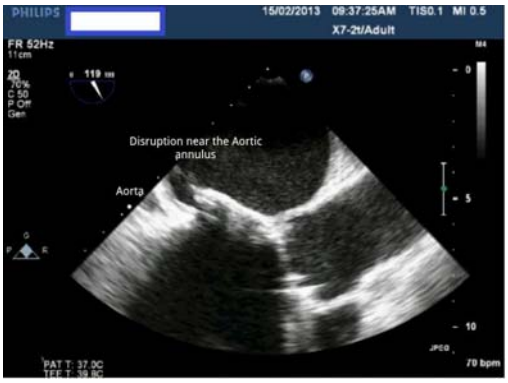
The 3D view using the 3D zoom facility of IE-33 Echo machine (iE33 MATRIX echo system, Philips, Netherlands) had given the exquisite and highly informative video of the prosthetic valve dehiscence affecting three-fourths of its circumference with the only attachment at the level of aortic root.

Reference:

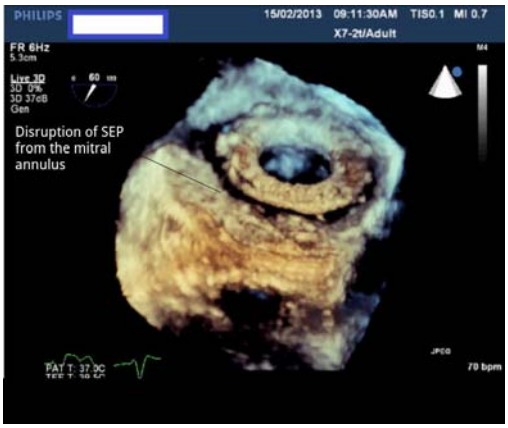
1) Foster GP, Isselbacher EM, Rose GA, et al. Accurate localization of mitral regurgitation defects using multiplane transesophageal echocardiography. Annals of Thoracic Surgery 1998;65:1025-1031



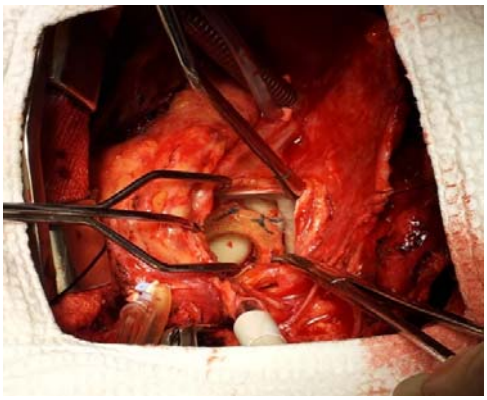
In the mid esophageal 2 chamber view, the SEP has dehisced near the inferior wall of left ventricle creating a large para-valvular space. In color flow Doppler, this space was filled by a large eccentric jet seen originating from outside the sewing ring.



In this modified 4 chamber view, the SEP in mitral position showed the characteristic rocking motion suggestive of dehiscence.



The 3D view of SEP using 3D zoom showing attachment of the valve only at the aortic root and dehiscence of three-fourths of the circumference of the sewing ring. The characteristic rocking motion is diagnostic of extensive prosthetic valve dehiscence.



Per-op confirmed the TEE reconstruction of the dehiscence; the valve was extruded and re-placed with a 31 St. Jude Masters™ (St. Jude Inc., Minneapolis, USA**)**

Iatrogenic LMCA Dissection

A 49 year old lady known case of bicuspid aortic valve with a gradient of 83/45 mmHg and moderate aortic regurgitation, had an EF of 42 %. The patient underwent a diagnostic coronary angiogram as per work up prior to elective aortic valve replacement.

During the angiogram Judkins Left 4 6F and Right 4 6F was used. During the angiogram the left catheter engaged non co-axially with the LMCA. The catheter jumped out of the LMCA ostia, the next angiogram showed dissection of the left main extending into the shaft.

The patient starting having angina in the cath lab, hence it was decided to stent the dissection, a 4 X 25 mm Angstrom II stent was deployed at 14 ATM to 4.26 mm, post stenting there was TIMI 3 flow in LAD and LCx territory. Echo showed severe LV dysfunction.

Following this the patient underwent a emergent aortic valve replacement with a 21 Chitra heart valve prosthesis and vein grafts to LAD and major OM branches, the LMCA ostia was closed from within the aorta to prevent further dissection.

Discussion:

Dissecting the coronaries whilst catheterisation is probably the complication most dreaded by interventionist, its incidence is pegged at 0.02- 0.07%[1, 3]. The lesion caused is usually picked up immediately once occurred but can go unnoticed[2].

Risk factors associated with dissecting the LMCA are calcification of the LMCA ostia or a calcific Aortic valve with calcification extending on to the ostia, atherosclerosis, complete total occlusion, anatomical variations in the take off of the LMCA from the Sinuses.

The mechanism most purported for dissection is bleeding into the vasa vasorum post intimal tear and intramedial hemorrhage, the intimal tear can happen while pressure driven expansion of a balloon or stent or the non coaxial engagement of the catheter in the ostia[4] what seems to have been the case in our patient.

The management of these patients is dictated by the course they follow post dissection and is a matter of debate and mandates a tailor made approach to such patient. The choice being between conservative management, percutaneous stenting or CABG[5]. Hemodynamic instability precludes a conservative approach[6].

The key factor of learning from our case was the bail out stenting to establish distal perfusion[6] and stabilize the patient hemodynamically. Since the patient had TIMI 3 flow post stenting CABG was undertaken[4] along with replacement of the aortic valve for which the patient had primarily come to us. After grafting the LAD with a vein, the dissection flap need to be addressed and was done so from within the Aorta by over-running it with 4-0 polyproplene.

It is probably prudent to subject these patients to surgery as the definitive modality of treatment as putting these patients on conservative management involves beta blockade and nitrates for myocardial protection and antithrombotic therapy, anticoagulation, antiplatelet

regimens which are imperative to reduce the potential of thrombosis in the false lumen and maintainflow in the true lumen, but this subjects the patient to a very real catastrophe of further disecction[7] and complete occlusion of the true lumen.

Prognosis was initially dismal with 50 % mortality and 50 % recurrence at two months. If the dissection is completely addressed the prognosis of these patients is good[7].

Reference:

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6) Lee SW, Hong MK, Kim YH. Bailout stenting for left main coronary artery dissection during catheter based procedure: Acute and long term results.Clin Cardiol 2004; 27: 393-5.
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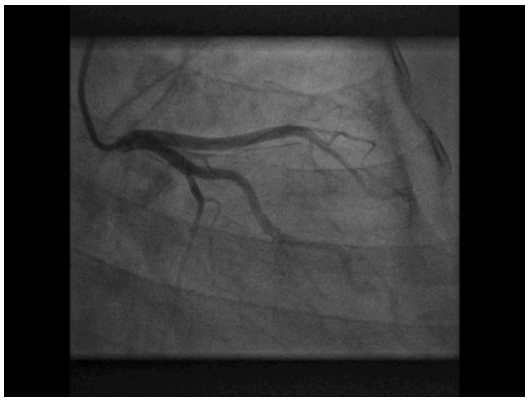

Figure 1: Non coaxial engagement of angiographic catheter

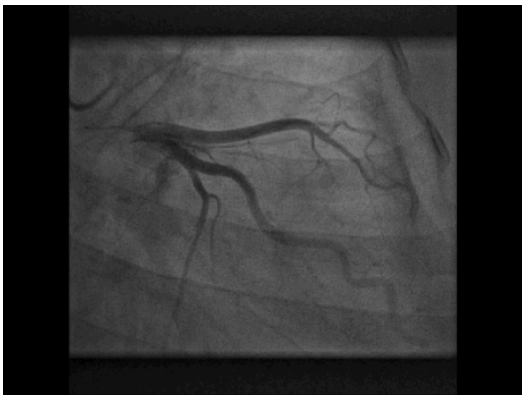

Figure 2: Displacement of the catheter

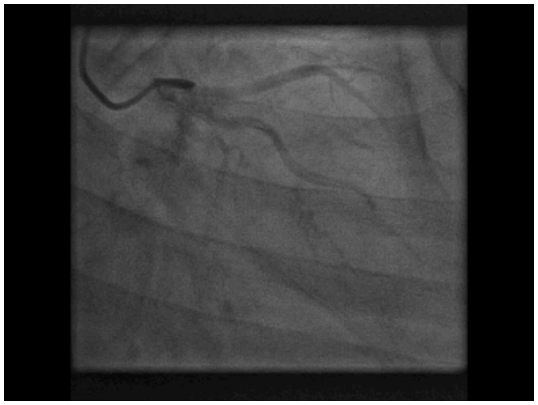

Figure 3: Dissection of the LMCA

Alfieri's Edge to Edge repair in a case of double inlet left ventricle (DILV) with moderate Atrio-ventricular valve regurgitation.

A 22 month old, 7 kg infant, diagnosed on antenatal scan to have a complex congenital heart disease, situs solitus, levo cardia, L-posed Aorta, slightly anterior to PA, Aorta arising from the rudimentary chamber and PA arising from the Anterior (LV) chamber. Moderate to severe Lt (tricuspid) AVVR, Rt AVVR mild. IAS intact. Planned for univentricular palliation.

The child was taken up for stage 1, BDG. As per institute policy we do BDG's on CPB.

The child was put on aorto-bicaval bypass with CPB time of 117 minutes and cross-clamp time of 52 minutes.

Table 1: Comparative pressures.

	Arterial	Pulmonary Artery	LAP	RAP
Pre-procedure	70/40/51m	19/12/16m	15/9/12m	8/4/6m
Post procedure	72/39/52m	42/20/29m	18/14/16m	18/12/14m

It was decided to do an Edge to edge repair (Picture 1). Alfieri stitch was taken using CV-7-0 on the leading edge of the AV valve, on water testing the repair, it seemed adequate and both the orifices allowed No 8 Hegar dilator. Post repair the PA pressure shot up to 42/20/29m and hence it was decided to band the PA's to mean pressures of 15 mmHg.

Post procedure the child had an uneventful recovery and epicardial ECHO, prior to discharge showed trivial central jet of regurgitation and no stenosis.

Discussion:

Atrio-ventricular valve regurgitation significantly alters the management of patients who are candidates for univentricular repairs and is an independent risk factor for early mortality[1, 2]. Prolonged exposure to the regurgitant flow predisposes the patient for pulmonary vascular disease, arrhythmias and congestive heart failure[2]. In patients with single ventricle physiology the atrioventricular valve is the gate keeper of theventricular function and the progression of the child to BDG or Fontan circulation, failing which the unique physiology deteriorates due to volume loading of the ventricle.

The very need to address the AV regurgitation is questioned, Imai et all[3] and Sallehuddin[4] and co-workers have showed an increase in operative mortality and Jae Gun Kwak[2] and associates did not find any benefit for early correction of the regurgitant valve but the patient showed better valvular function at midterm.

Another issue of much debate is the timing of the repair, most prefer to repair the valve at time of BDG or between stage II and III, earlier the repair would amount to better preservation of ventricular function as it would take the volume load of the single ventricle

situation which is much different than a two ventricle situation[6] which perpetuates less incidence of CHF, arrhythmias, pulmonary vascular disease. These patients are to be taken along the BDG and Fontan pathway as early as possible to reduce the volume overload. Hancock Friesen et all suggested that if the degree of AVVR is mild prior to stage II, some improvement is expected after volume off loading of the stage II procedure[7] and the same can be extrapolated to stage II-III interim.

The procedure of repairing the valve is dictated by the morphology of the same be it annular dilation, prolapsed, dysplasia, cleft, chordal elongation of restriction or a combination of these.

Repair techniques described include annuloplasty (localised or annlar), commissuroplasty, suture repair of dysplastic leaflets, edge to edge repair and closure of the cleft with or without annuloplasty or commissuroplasty and artificial valve replacement[2, 5], which is considered the last option[2].

In situation where there is flail leftlets to allow for a little more of tissue handling, the edge to edge repair is the preferred method[7].

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7) Makoto Ando MD and Yukihiro Takahashi MD. Edge-to-Edge Repair of Common Atrioventricular or Tricuspid Valve in Patients With Functionally Single Ventricle. Ann Thorac Surg 2007;84:1571-1577.

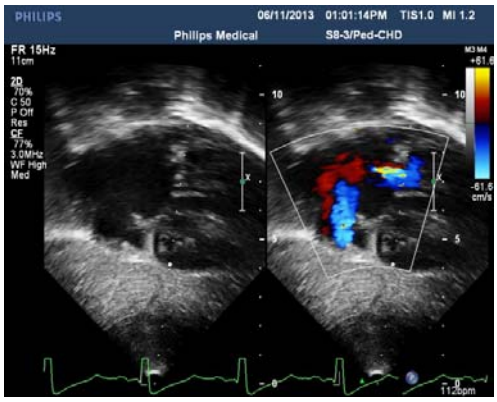

Figure 1: Preop ECHO showing mod-sev LAVVR

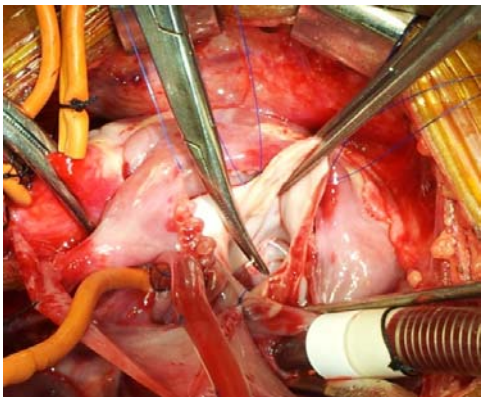

Figure 2: Perop picture showing Alfieri's sticth

Differential saturations in 4 limbs, an unusual presentation

Case:

A infant weighing 3.4 Kg was detected to have anomalous origin of RPA from ascending aorta, the MPA continued as LPA, PDA was shunting right to left, the child had suprasystemic RV pressure and flow reversal in the descending thoracic aorta. The lower limb saturations were 55 % and left upper limb saturations were 73 % and right upper limb saturations were 84 %.

Discussion:

For understanding this concept, the aorta can be divided into 3 segments, from the valve to the origin of the RPA, from RPA to the insertion of the PDA and the third segment being from insertion of PDA onwards. The right lung was getting it supply from the systemic run off via the ascending aorta and the left lung was being supplied by the right ventricle, due to the severely raised Rp, the child was shunting right to left shunt at the level of the PDA causing preferential flow of desaturated blood maximally in the descending thoracic aorta. The flow reversal in the aorta during diastole caused it to flow back into the arch and ascending aorta. Due to the run of into the RPA this flow reversal was augmented resulting in sequential lower saturations in the left and right upper limbs.

The presence of differential cyanosis in children is almost pathognomonic of certain physiologies, upper limb saturations being more than lower limb saturations occurs more commonly and is seen in PDA with preductal CoA or Arch hypoplasia, PDA with transitional or persistent pulmonary hypertension of new born.
Reverse differential cyanosis is seen in cases when the right to left shunt is preferentially being diverted to the cephalad circulation as opposed to the more common caudad circulation due to stenosis and is almost diagnostic of TGA with pre ductal Coa or interrupted aortic arch, TGA with PDA and PAH.

When viewed critically this difference in cyanosis can pick up congenital heart disease in apparently asymptomatic neonates[1]. Rarely cyanosis presenting selectively in right upper limb can clinch the diagnosis of a right aberrant subclavian artery arising from the DTA in a patient of PDA with pulmonary hypertension, the child will present with cyanosis of lower limbs along with right upper limb. Rarely will there be a presence of bilateral PDA[2], the right continuing as subclavian artery. Streaming also cause reverse differential cyanosis as in occurs in supracardiac TAPVC[3].

In a patient with severe pulmonary hypertension bordering on Eisenmenger's syndrome, differential cyanosis along with differential clubbing points to the PDA as the sole diagnosis[4,5].

Hence it is imperative to check the saturations in all 4 limbs of infants as part of cardiovascular workup and a difference in the saturations can explain the physiology just as well as absolute cyanosis.

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4) Rachel Wald MD, Andrew Crean MRCP. Differential clubbing and cyanosis in a patient with pulmonary hypertension. CMAJ June 2010 182(9)
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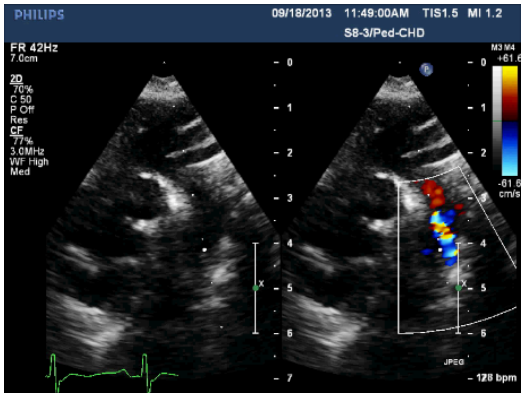

Figure 1: Colour compare image on flow reversal in proximal descending aorta.

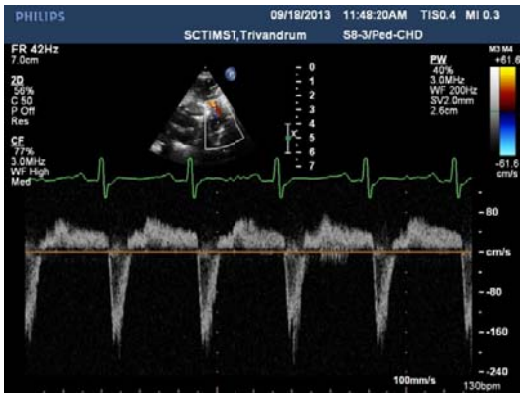

Figure 2: Pulse wave Doppler with diastolic flow reversal in descending aorta.

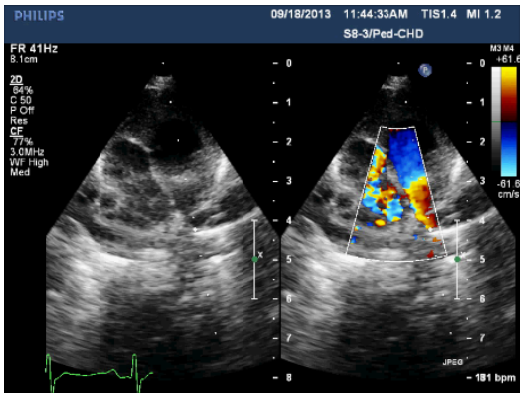

Figure 3: Colour compare image showing the origin of the RPA from aorta and MPA continuing as LPA.

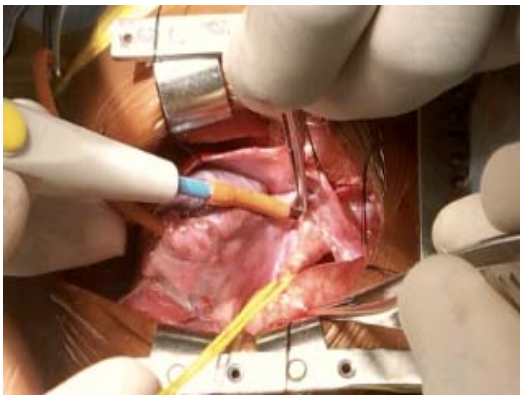

Figure 4: Peroperative picture showing the RPA looped at its origin.

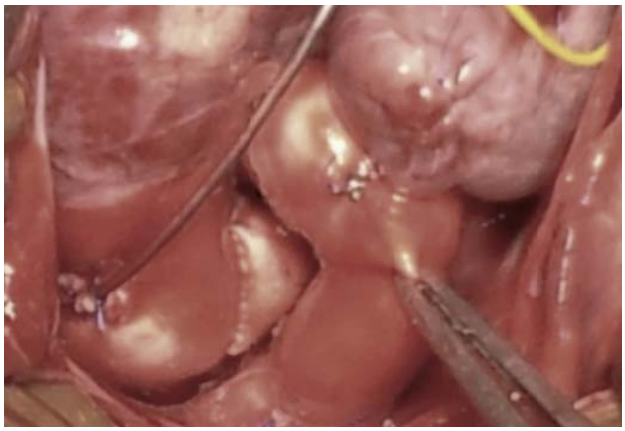

Figure 5: Perop picture of reimplanted RPA to MPA.

Modified BT shunt post iatrogenic injury following attempted PDA stenting

2 patients who underwent RMBTS post iatrogenic injury following attempted PDA stenting.

Case 1:

A 12 day old neonate weighing 2 kgs, diagnosed as pulmonary atresia, intact ventricular septum with duct dependent pulmonary circulation, post PDA stenting with suspected spasm/thrombosis of the PDA was taken up for BT shunt.

PDA stenting was attempted via Rt axillry, the PDA was taking off vertically from the under surface of the Arch just opposite to the LSCA, after the 1st unsuccessful attempt, the 2nd attempt at crossing the PDA resulted in dissection of the LSCA hence bailed out.

The child was taken up for BT shunt with borderline hemodynamics, saturations of 60% on adrenaline of 0.1 mics.

Per-op the child had a hematoma extending from root of neck into superior mediastinum, involving Ascending Aorta to beyond the origin of PDA. What was suspected spasm turned out to be transaction of the LSCA at its origin with active bleed. The PDA stent was extending into Right pulmonary artery. There was an intraluminal shelf opposite insertion of PDA on the LPA causing stenosis.

Since the bleeding obscured the field, the surgery proceeded on total circulatory arrest at 18° C, after clamping Arch vessels, patient was drained. Both ends of LSCA over sewn with 7-0 prolene. MPA opened, stent extruded and LPA plasty done with bovine pericardium without dividing the intraluminal shelf. Rewarming started, deairing achieved and Aorta declamped. The RMBTS proceeded in a standard fashion with a 3 mm ePTFE tube graft anastomosed to Innominate artery and RPA using 8-0 prolene, child shifted to ICU with the sternum stented electively and on adrenaline, nor-adrenaline and vasopressin, PD was inserted and the child was maintaining fair hemodynamics with a balanced circulation situation.

The child was taken up for delayed sternal closure 2 days later, immediately following closure the child arrested, the sternum was reopened and cardiac massage given but the child could not be revived.

Case 2:

1 day old neonate weighing 2.7 kgs, diagnosed as pulmonary atresia, VSD, d-TGA, PDA dependent pulmonary circulation, p/dissection of DTA following attempted PDA stenting.

The PDA stenting was attempted via the right Axillary artery, Arch angio showed a vertical ductus suppling confluent pulmonary arteries, multiple attempts to negotiate the ductus were unsuccessful due to aberrant RSLC which was taking origin posterior to the Aorta at the level of the ductus. During one attempt the DTA dissected and the procedure was abandoned.

Post procedure showed laminar flow in DTA, neck vessels free of flap. The child was maintained on prostaglandin infusion and saturation maintained. CT done after stabilizing the child showed type III dissection extending from distal Aortic arch to abdominal aorta.

The child was taken up for RMBTS, the procedure proceeded in a standard fashion using a 3 mm ePTFE tube graft suture to innominate artery and RPA using 8-0 prolene. The childs post op recovery was uneventful and the was discharged after 8 days. Prior to discharge the ECHO showed functioning BT shunt and the dissection status quo.

Discussion:

The current trend is towards stenting the PDA instead of doing a palliative shunt. No doubt the stenting would be a wiser choice in some patients like wt of less than 2000 gm, small pulmonary arteries, McGoon ratio of less than 1.5 and it also off sets the need for an additional procedure[1]. This rule however cannot be applied as a blanket cover to all PDA's.

The choice of stenting the ductus or doing a MBTS is a matter of constant debate.

Patients probably not suitable for stenting are those with a tortuous and long PDA's, unrestrictive PDA's ie > 2.5 mm at their narrowest point, presence of branch PA stenosis and the origin of the PDA on the Aorta[1]. Accordingly the PDA classified as

Type I: PDA takes origin from Aorta distal to SCA

Type II: Origin from underneath the Aortic arch in between LSCA and LCCA

Type III: Origin from underneath the Aortic arch proximal to LCCA

Type IV: Origin from innominate/SCA.

The course of the PDA is another important aspect to consider, whether it is tortuous, sigmoid, vertical, short and with a wide ampulla.

The presence or absence of intra luminal stenosis of PA's prevents engagement of the stent and also causes branch PA stenosis.

It happens not so infrequently that even though the patient underwent failed stenting (case 1) or stenting following which the stent thrombosed and failed attempts at thombolysis or clot disruption[2] (case 2). Taking up these patients for a surgical procedure becomes a bit trickier.

Since there are no controlled trials addressing the issue of choice between the two procedure data is limited to case series and individual observations[3].

Surgery is warranted in patients with truly duct dependent (i.e no other source of pulmonary blood flow) because ducts are known to go into spasm with overzealeous handling or spontaneously, pushing the neonate to the edge. The spasm can affect duct of any size and larger ducts are not immune and it would be a folly for a novice angiographer to expect otherwise.

Patients of pulmonary atresia have PDA's which have a tortuous morphology, hence these patients would benefit from defining the anatomy of the ductus further, Lin et all have reported the use of multi-detector CT to assess these ducts for stenting[5] and stent related complication, in our second case CT scan done post PDA stenting showed a PDA which was sigmoid and tortuous and also did define the extent of dissection.

Reference:

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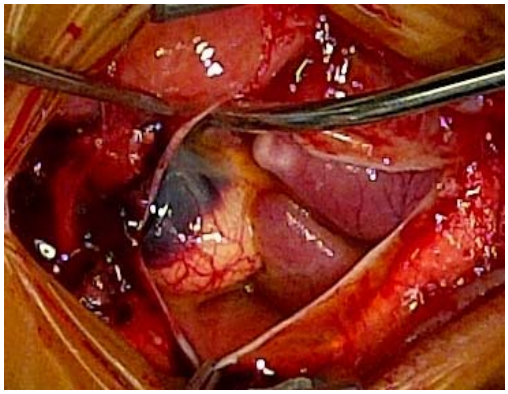
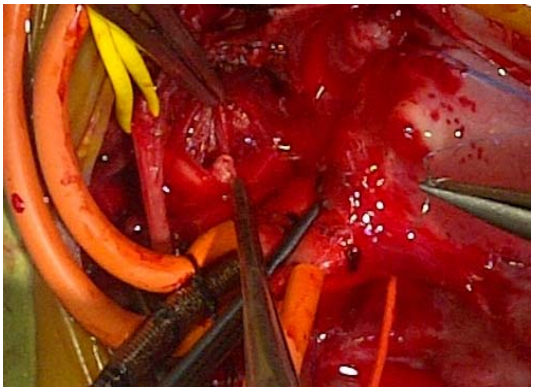
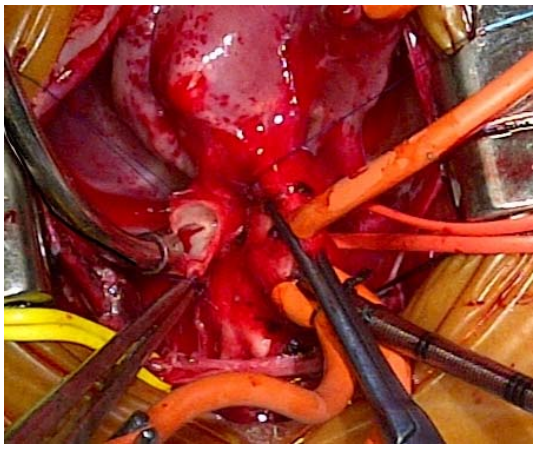
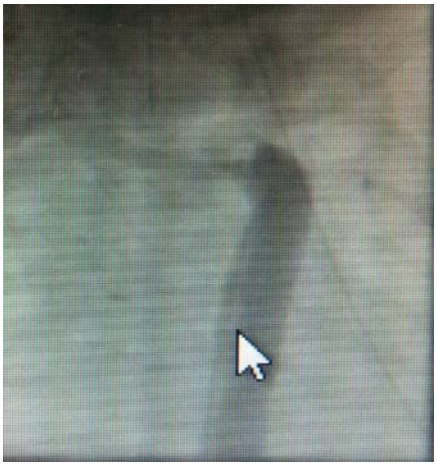




Case 1: Avulsion of LSCA from Aorta causing hematoma over the neck vessels.



Shelf in the LPA which prevented the engagement of stent.



Case 2: Dissected DTA

Nightmare session

A 4 year old girl with simple ASD was taken up for surgical closure of ASD via ministernotomy. A 6 cm incision was placed below the nipple line and the lower sternum divided. The aorta was cannulated with 16 Fr Fem-Flex II canula and bicaval canulation with two separate venous lines to the reservoir. As the venous canulae chosen was two sizes below the recommended, the venous drainage was slightly inadequate hence a vacuum assist for venous drainage was instituted. The venous drainage improved immediately.

As preparations were being made for the cardioplegia, the perfusionist noted a decrease in venous return and called out. The next second the IVC line had an air lock which was clamped and immediately taken out. When the IVC pursestring was loosened the heart made that unmistakable sound and air gushed out of the IVC pursestring. The next moment the SVC line also had an airlock which was clamped and removed. The ministernotomy was converted into full sternotomy and suction bypass started. Air bubbles continued to escape from both caval pursestrings. RA appendage pursestring was taken, and a bard canula inserted, when the SVC line clamp was removed to connect to the Bard canula the pathology was revealed, a mixture of air and blood came spraying out of the line. We realized we had a pressurized venous reservoir. By this time the heart had become flaccid and dark, air had gone into the coronaries and the systemic circulation.

The VAVD was removed, bypass re-established, patient cooled. Retrograde cerebral perfusion done. Postoperatively patient was ventilated for a day. Patient made a complete recovery with no neurological deficits.

All this had taken place because the indicator needle of the vacuum regulator was stuck.

This case of venous air embolism going over to the systemic circulation that was successfully managed and patient made an uneventful recovery. This is not just a nightmare that can occur in dreams but daymare that can occur in routine Operation theatre.

Original Papers

1) Demographic Profile, Clinical Characteristics and outcomes of patients undergoing Coronary artery Bypass grafting-Retrospective analysis of 4024 patients -Are Indian patients different?

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