



Billroth
Hospitals

DEPARTMENT OF CARDIOLOGY

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CASE REPORT - 1

A CHALLENGING CASE OF SUCCESSFUL SURGICAL MANAGEMENT OF **HOCM** IN A **YOUNG FEMALE.**



A 24 year female, diagnosed with hypertrophic obstructive cardiomyopathy in 2018, on medical management presented to our hospital with complaints of dyspnoea on exertion NYHA III, giddiness and palpitation since 2 weeks. Her ECG showed signs of left ventricular hypertrophy and significant q waves in leads II, III and aVF. Two dimensional ECHO revealed significant left ventricular outflow obstruction during systole with peak gradient of 93mm Hg and mean gradient of 60mm Hg across the LVOT, systolic anterior motion of anterior Mitral leaflet with moderate Mitral Regurgitation, no regional wall motion abnormalities and normal LV function.

A subsequent cardiac MRI confirmed asymmetric hypertrophic of LV with significant LVOT obstruction and few mild patchy fibrosis of LV muscle. Following this, she was taken up for trans-aortic septal myectomy (Morrow's procedure)

Intra-operatively, after placing the patient on cardiopulmonary bypass. Aorta was cross clamped and heart arrested with cold Del Nido cardioplegia. Transverse aortotomy was done and hypertrophied sub aortic septum identified. 1.5 x 1 x 2 cm of septal myocardium excised taking care not to injure the membranous septum. Around 6-8 gram of septal tissue was excised Aortotomy was closed, heart deaired and aortic cross clamp removed. Heart picked up in normal sinus rhythm following which heart was weaned off cardiopulmonary bypass. Patient was shifted to CTICU with normal hemodynamics and was extubated after few hours. First post operative ECG was noted to have complete LBBB which documentary evidence of adequate LVOT obstruction relief as we remove the tissue along with left bundle. She made a gradual recovery over the next few days and 2as discharged on 5th post operative day.

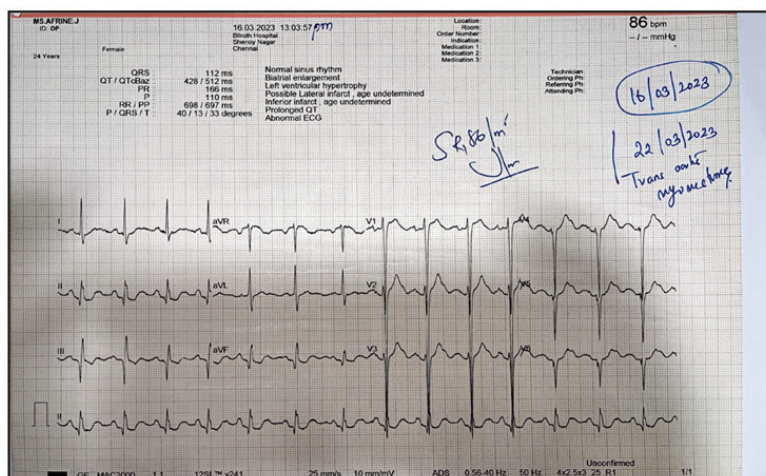
Her post operative 2D ECHO showed a maximal LVOT gradient of 30 mm Hg and no systolic anterior motion of AML with trivial mitral regurgitation. On Second post operative review, she was asymptomatic and her LVOT gradient was noted to be 0. She was asymptomatic till recent follow up. Histopathology report typical Hypertrophic cardiomyopathy pattern noted myofibre disarray, muscle hypertrophy, interstitial and pericvascular fibrosis

BACKGROUND

Hypertrophic obstructive cardiomyopathy (HOCM) is the commonest cardiac genetic disorder. HOCM is a significant cause of sudden cardiac death in young people, including well-trained athletes, affecting men and women equally across all races. In most patients, it results from asymmetric interventricular septal hypertrophy, causing outflow obstruction of the left ventricle. Diagnosing this condition is a challenge since many a times the first symptom is a major cardiac event. We present a case of HOCM in a young symptomatic female who was treated successfully through surgical management.

DISCUSSION

Hypertrophied cardiomyopathy is a genetic disorder characterized by hypertrophy of the heart in the absence of pressure overload. HCM is due to missense mutation of genes that encode for beta myosin heavy chain of cardiac sarcomere proteins. It is transmitted as an autosomal dominant trait. The prevalence of HCM in the general population worldwide is 0.2% (1 in 500 adults), as determined by echocardiographic studies(1). The cardiac myocytes are hypertrophied, disorganized and separated by areas of interstitial fibrosis. Characteristic cardiac structural changes include increased left ventricular wall thickness causing dynamic left ventricular outflow obstruction, diastolic dysfunction, myocardial ischemia, arrhythmias, autonomic dysfunction, and mitral regurgitation.

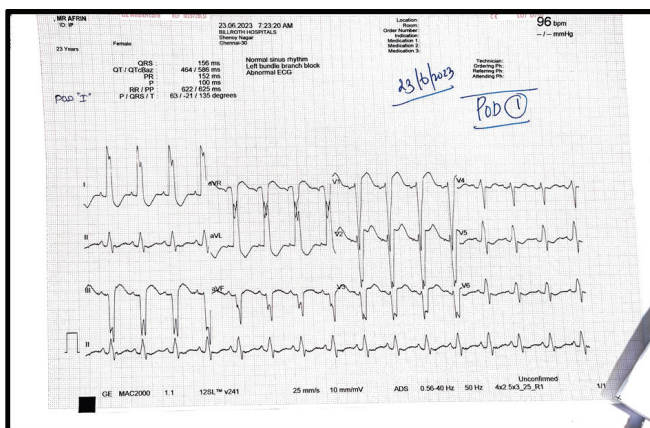


Pre operative ECG

HOCM can be classified as obstructive or non-obstructive. There is some degree of asymmetric left ventricular hypertrophy. The degree of obstruction and clinical presentation depends on the extent of hypertrophy. It most commonly affects the ventricular septum (about 2/3 of patients), although any portion of the left ventricle can be affected. Dynamic outflow obstruction in HOCM is due to the systolic anterior motion of the anterior leaflet of the mitral valve. This is due to impingement of the mitral valve leaflets on the hypertrophied basal septum. The outflow tract obstruction is dynamic and caused by a pressure gradient, which pulls the anterior leaflet of the mitral valve anteriorly, further leading to outflow tract



obstruction. The degree of obstruction is dependent upon contractility and loading conditions. In 25% of patients, the obstruction can exist at rest, but in about 70% of patients, it can be brought about with provocative maneuvers. Most patients with HOCM have an abnormal diastolic function. This increases left ventricular pressures, impairing ventricular filling, which further exacerbates obstruction. Because the coronary vessels are filled during diastole, in combination with outflow tract obstruction and ventricular stiffness, there is an increased risk for myocardial ischemia. This may be responsible for ventricular arrhythmias and sudden death. In severe cases, this can occur at rest. More commonly, it occurs with provocative maneuvers such as exercise during increased myocardial demand (2)



First post operative ECG

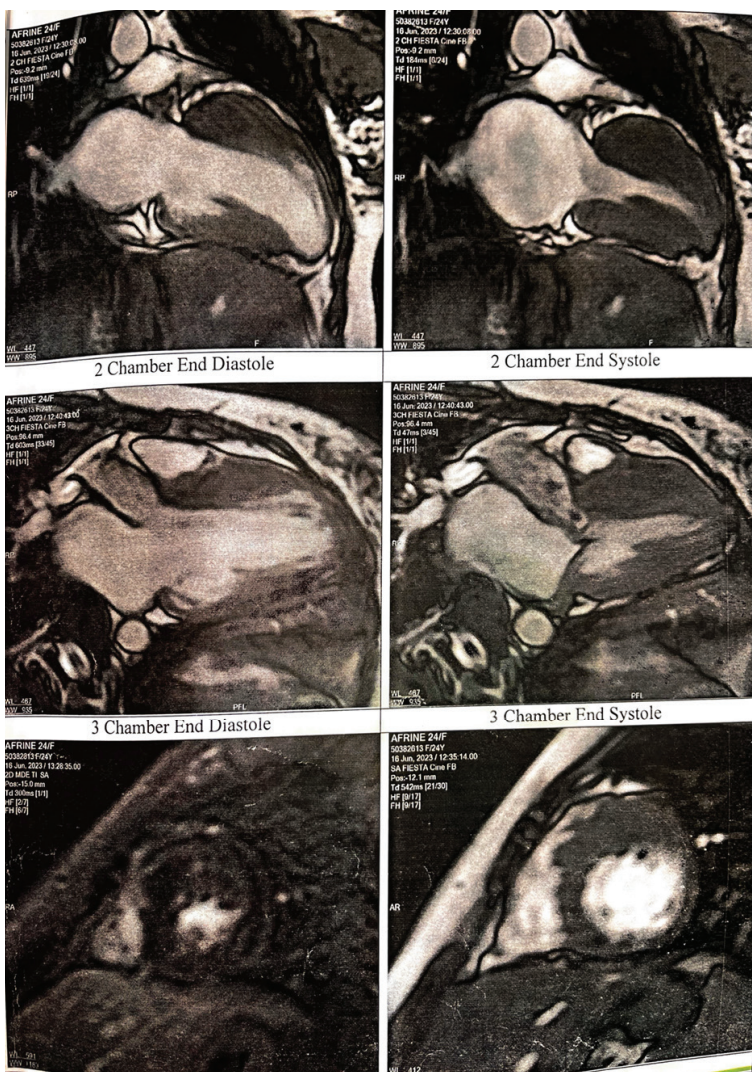
HISTOPATHOLOGY TEST (SMALL)	
Brief Clinical History:	
Hypertrophic obstructive cardiomyopathy (HOCM).	
Specimen:	
Biopsy from hypertrophied septal muscle	
Macroscopic Description:	
Received multiple irregular greyish tan muscular tissue bit measuring 0.3 cm to 1.5 cm in greatest dimension.	
A, B - all.	
(Dr.GR).	
Microscopic Description:	
A, B : Sections show myocardial tissue with haphazard arrangement of fibres with disarray, interstitial and perivascular fibrosis highlighted by Masson's trichrome stain. Some hypertrophic muscle fibres seen. No significant inflammation identified. Congo red stain is negative for amyloid deposition. No evidence of vascular thickening or thrombi seen.	
IMPRESSION:	
HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY, SEPTAL MUSCLES, EXCISED SHOWING CARDIAC MYOCYTE DISARRAY, INTERSTITIAL AND PERIVASCULAR FIBROSIS AND SOME HYPERTROPHY.	
Comments:	

HPE report

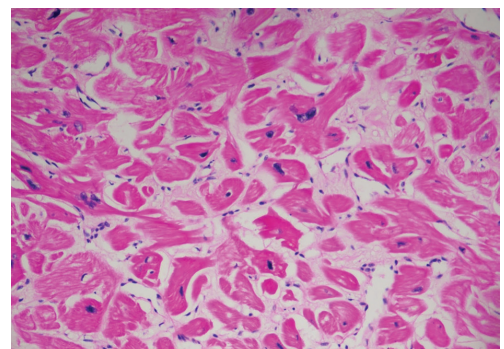
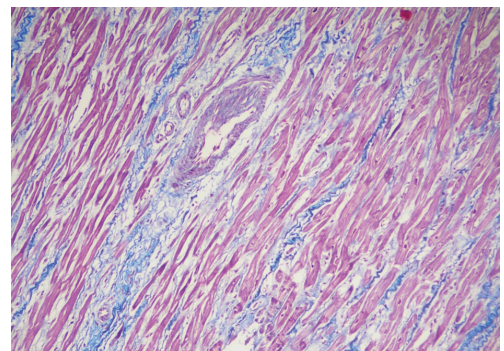
The involvement of the mitral valve in HOCM was first detected when M-mode echocardiography was introduced and the SAM became evident. Over years this phenomenon is attributed to a Venturi effect. It is, however, due to an initial systolic flow deviation by the bulging septum towards the mitral valve pushing the septal leaflet anteriorly. It makes therefore sense that concomitant mitral insufficiency will disappear once the septal bulge is resected. To achieve complete resection is not easy and surgery should be performed in specialized centers by experienced surgeons. The transaortic access is standard nowadays but visibility to the lower end of the septal bulge is highly restricted (3) In case of mitral valve regurgitation due to multiple pathologies including anteriorly directed MR jet, flail chordae etc, mitral valve replacement should also be considered along with septal myectomy.



Septal myectomy should be considered for any patient who remains symptomatic (NYHA class III or IV) after appropriate medical therapy (β -adrenergic receptor blockade, calcium antagonists, disopyramide), failed septal ablation, failed pacemaker therapy, LV subaortic gradient (≥ 50 mmHg) at rest or after physiologically based provocation with exercise, onset of atrial fibrillation

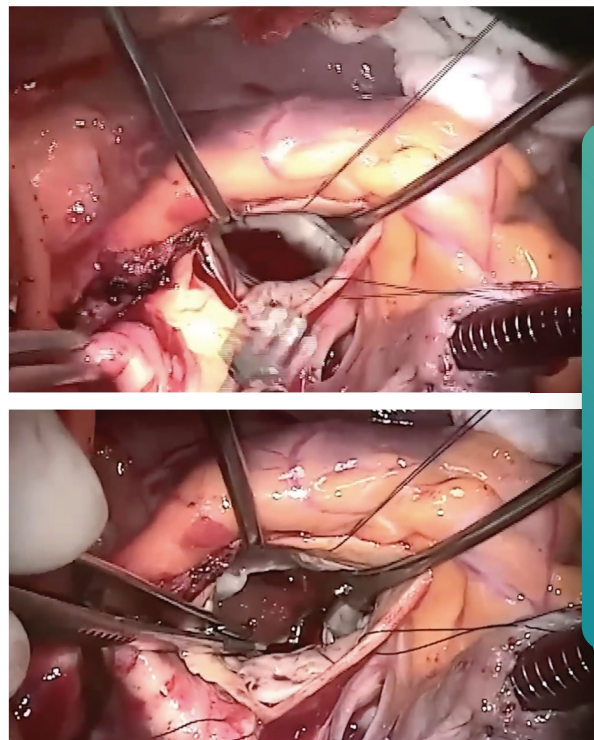
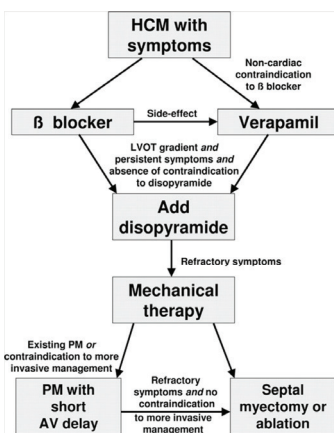
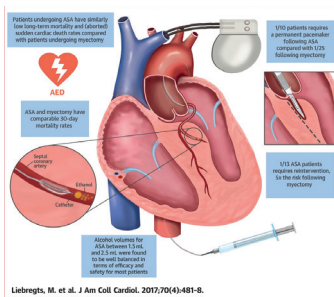


Common post-operative complications include residual gradient due to incomplete resection of septal myocardium, left bundle branch block, right bundle branch block or complete heart block requiring permanent pacemaker. In their paper Hong et al. indicate that postoperative heart block requiring permanent pacemaker had no influence on overall mortality.(4)





Surgical myectomy performed to relieve outflow obstruction and severe symptoms in HCM was associated with long-term survival equivalent to that of the general population, and superior to obstructive HCM without operation. In this retrospective study, septal myectomy seems to reduce mortality risk in severely symptomatic patients with obstructive HCM.(5)



Finally, cardiac transplantation should be considered for suitable candidates with HOCM who have not responded to maximal medical and surgical therapy. These patients usually have intractable symptoms of heart failure associated with dilated ventricular cavities.

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Steve R Ommen 1, Barry J Maron, Iacopo Olivetto, Martin S Maron, Franco Cecchi, et al; Long-term effects of surgical septal myectomy on survival in patients with obstructive hypertrophic cardiomyopathy

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CASE REPORT - 2

A RARE CASE OF HEART FAILURE – CAVOPULMONARY WINDOW TREATED BY PERCUTANEOUS INTERVENTION.

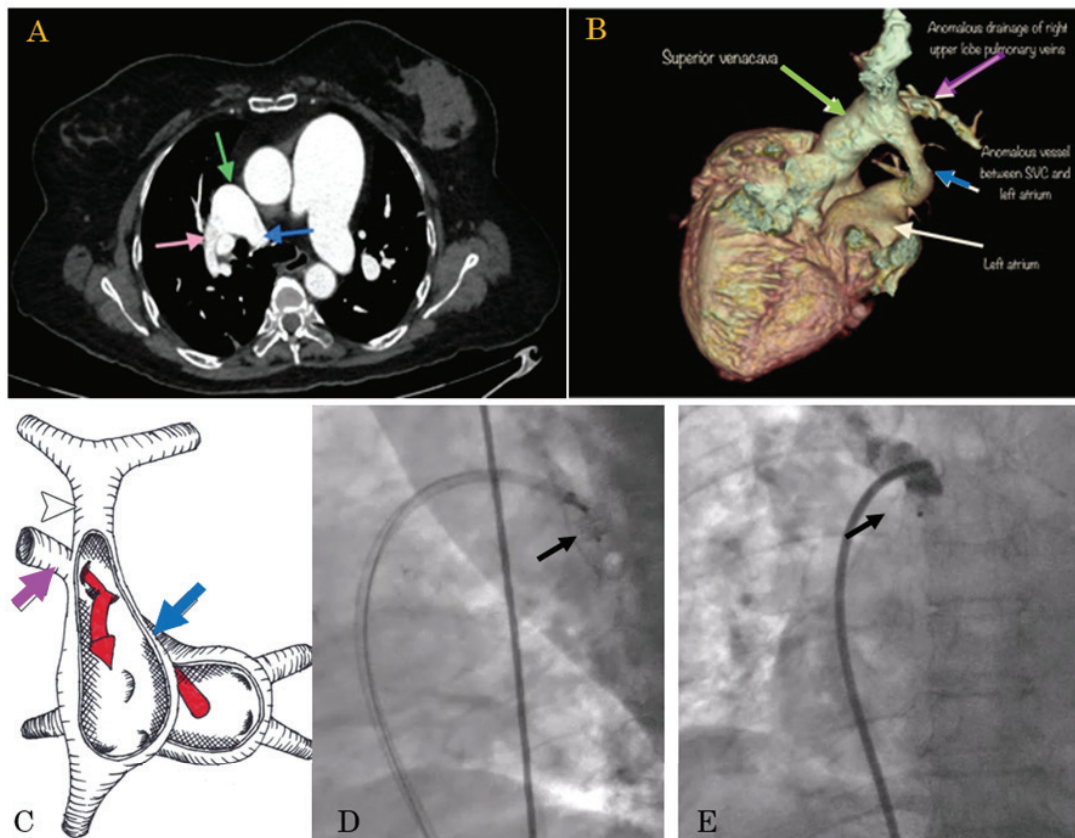


65 year old female, diabetic and hypertensive for 6 years presented with severe leg swelling for 1 month with oozing of fluids from both legs with skin redness involving both legs past 1 week. History of breathlessness on exertion for past 3 to 4 years with history of orthopnea for past 1 year. Initially able to sleep with 2 to 3 pillows on right lateral side. Now past few months sleeps in sitting position only. She was found to be tachypneic with saturation of 88% and had anasarca and lung crepitations. ECG showed atrial flutter with 3:1 block. Echo showed a D - shaped left ventricle with adequate systolic function with moderate LV diastolic dysfunction, dilated pulmonary artery (PA) and dilated right atrium (RA) and right ventricle (RV), with RV dysfunction. ECHO also showed Severe TR with estimated pulmonary artery (PA) pressure of 70mmHg. No evidence of DVT in Lower limb Doppler study. We did CT-chest with contrast with an intention to assess the pulmonary parenchymal as well as any vascular lesions. This revealed an anomalous connection between left upper pulmonary vein (LUPV) and superior vena cava (SVC) and an anomalous connection between the SVC and the left atrium (LA). Based on CT findings a diagnosis of **cavopulmonary window / bi-atrial drainage of SVC was made.**

She was managed with diuretics, digoxin and treated for lower limb cellulitis and iron deficiency. After 1 week was discharged with Torsemide 20mg, Spironolactone 50mg, and Rivaroxaban 15 mg along with Tadalafil 20mg and Ambrisentan 5 mg. Plan was to do catheterization after stabilization. Patient was willing for cath study but was not willing for surgical intervention. After two months of initial presentation patient was taken up cath study with a plan of percutaneous intervention, if feasible. The study revealed a significant left to right shunt with normal pulmonary vascular resistance with mild pulmonary hypertension and there was no significant change in the right and left heart filling pressures with balloon occlusion test. So after discussing the surgical option again which was turned down by the patient, we decided to proceed with device occlusion of the anomalous SVC to LA connection. The connection was approached from right femoral vein and through SVC into the anomalous LA connection a 10 mm Amplatzer vascular plug II device was deployed successfully. Patient tolerated the procedure well and was discharged after two days



Now on 8 months follow up, the patient is in functional class II with no signs of volume overload and patient is able to sleep in supine position. Her estimated PA pressure has reduced from 70mmHg to 45 mmHg with normal RV function. However the severe tricuspid regurgitation is persisting. She is continued on low dose diuretics and Rivaroxaban 15mg.

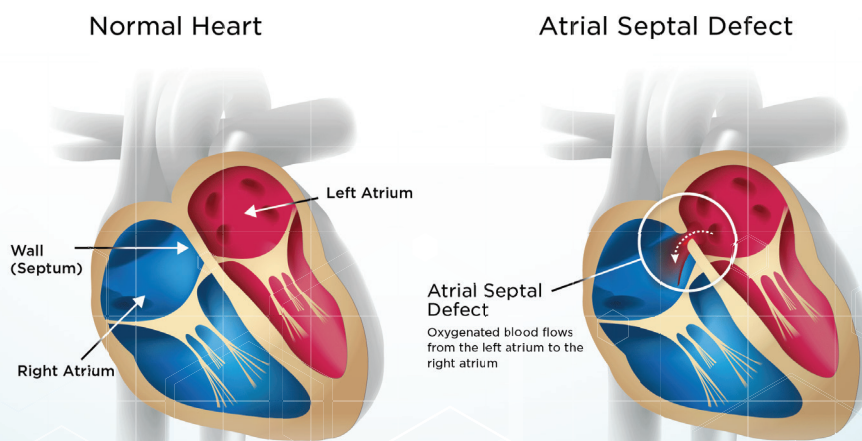


Cavopulmonary window or Veno-venous Bridge or Bi-atrial drainage of SVC is an extremely rare condition. So far only 8 cases have been reported in literature, of which only three have been operated and one underwent covered stent deployment for SVC. Most presentation is similar to ASD with left to right shunt with volume overloaded right atrium and ventricle, except when there is stenosis or atresia of SVC to RA junction where the presentation is in early infancy with cyanosis. Surgical correction of the anomaly is the preferred mode of treatment when possible.

TREAT-TO-CLOSE DEVICE CLOSURE OF ASD IN AN OCTAGENARIAN WITH ASD & SEVERE PH



Introduction: Atrial septal defect is one of the common congenital heart diseases (CHDs) affecting nearly 10-15% of all CHDs. It has a birth prevalence of 1.6-2.5/1000 live births, 25-30% of which present in adulthood. Majority of the patients with ASD have no or mild restriction of physical activity and paucity of clinical findings. Hence diagnosis may be delayed until late in life. As many as 70% of ASDs were diagnosed in 4-5th decades in the past. But with the widespread use of echocardiography for various indications like routine checkups prior to employment or during pregnancy, earlier diagnosis is made possible in asymptomatic patients. Being a pre-tricuspid shunt, pulmonary hypertension (PH) is uncommon, affecting 9-22% of them, mostly occurring in 4th decade of life. Progressive PH leads to Eisenmenger syndrome manifesting bidirectional or right to left shunt, right ventricular (PH) dysfunction, RV failure and death. Atrial arrhythmias like atrial fibrillation and stroke due to paradoxical embolism are the other morbid events that may happen in late adulthood. Such worse outcomes can be prevented by closure of ASD either by surgery or device early in life, particularly before the age of 30 years. The presence of severe PH is generally regarded as a contraindication for surgical or device closure. This is true when Eisenmenger syndrome develops and there is reversal of shunt. But when there is evidence of predominant left to right shunt, reduction of Pulmonary Artery (PA) pressure by drug therapy may allow these patients with severe PH to safely undergo defect closure. We present one such case of device closure of ASD in an octogenarian in whom PH was so severe that defect closure was initially considered to be futile but was made possible by suitable drug therapy prior to closure.





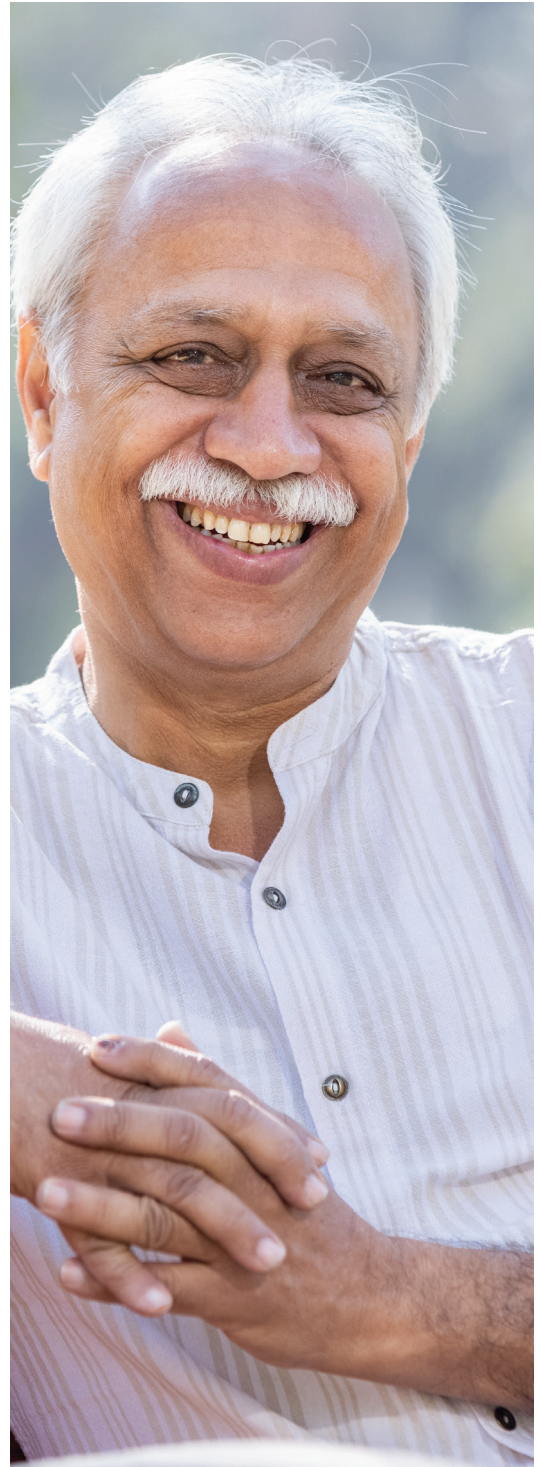
Case Report: Mr. S.R 79 years, presented with class III dyspnea of 3 months duration, in January 2020. He was on medications for COPD, Hypertension and CAD. A Coronary angiogram done in the year 2017 revealed calcified coronaries with mild obstructive disease. Physical examination revealed a pulse rate of 98/min, BP of 124/90 mm Hg, SPO₂ of 92%, elevated JVP, mild leg edema, widely split S₂, 2/6 ejection systolic murmur over left II ICS and 2/6 holosystolic murmur over left IC ICS. 6-minute walk distance was 150 meters with reduction of SPO₂ from 92 to 88%. ECG revealed sinus rhythm, Right axis deviation, Right IVCD and ST-T changes in anterior leads. Chest X Ray showed a CTR of 60%, enlarged pulmonary arteries and pruning of peripheral branches suggestive of advanced PH. Echocardiography showed right and right ventricular enlargement, right ventricular hypertrophy, mild RV dysfunction, large fossa ovalis type of ASD (26 mm) with left to right shunt and severe PH (estimated PA systolic pressure 76 mm Hg), moderate mitral regurgitation and normal LV function.

Having thought that he was in a state of advanced PH, he was prescribed Sildenafil 25 mg tid and mentioned that his heart defect was beyond correction. He was reevaluated a month later and to our surprise his functional capacity improved with a 6-minute walk distance of 280 meters, SPO₂ of 98%, normal JVP and reduced PA systolic pressure to 45 mm Hg by echo. Hoping to lower the pulmonary artery pressure further, Ambrisentan 10 mg od was added. Reassessment was done again in August 2020 which revealed a PA systolic pressure of 42 mm Hg. Since he was reluctant to undergo TEE for deciding suitability of device closure, contrast CT was done which revealed adequate rims except a smaller retro aortic rim. He was then advised to undergo right heart catheterization (RHC) and evaluate the reversibility of PH followed by device closure of the defect, if hemodynamics is suitable. It was done in December 2020. PA systolic pressure was 44 mm Hg (mean 27 mm Hg), with a11% O₂ step-up at atrial level. PVR was 1.8 Wood units, PVR/SVR ratio was 0.11 and Q_p/Q_s was 1.7. Since PH was mild at the time of RHC, reversibility study was not performed. Coronary angiography revealed occluded circumflex artery with mild disease of left anterior descending and right coronary arteries. Under fluoroscopic and echocardiographic guidance, a 36 mm Amplatzer device was placed across the defect and hemodynamics was observed for 30 minutes. PA systolic pressure rose to 45 to 54 mm Hg and LVEDP rose from 17 to 25 mm Hg. Since he clinically tolerated the procedure, the device was released and he made an uneventful recovery.



He is being followed up with serial echocardiographic studies which showed that moderate MR continues to be present with PA systolic pressure being maintained between 45 to 55 mm Hg. Sildenafil and Ambrisentan are being continued. He continues to be in functional class II and his current 6-minute walk distance is 300 meters.

Message: This case report highlights the benefit of aggressive management of CHDs in adults. Though closure of ASD with PH in elderly was originally considered harmful or not necessary, there is now ample evidence that closure of the defect, can still offer benefit in terms of improvement of symptoms and functional capacity. Of course, it may not prevent atrial arrhythmias that are common in adults with ASD. The currently available PH specific therapies that include PDE5 inhibitors like Sildenafil, Tadalafil; Endothelin receptor antagonists like Bosentan, Ambrisentan and Macitentan; soluble Guanylate cyclase stimulator like Riociguat and Prostacyclin analogues like Epoprostenol, Treprostinil and Prostacyclin receptor agonist namely Selexipag have revolutionized the management of PH both idiopathic and secondary forms like CHD with PH. These drugs improve the exercise capacity and prolong survival in idiopathic PH patients. The mere presence of severe PH should not be considered as a contra indication for closure of congenital heart defects, particularly ASDs. Administration of PH specific drugs for 3-6 months and re-evaluation may be done in all, preferably with cath studies to decide the suitability. A systematic review and meta-analysis of 18 cohorts comprising 1184 patients suggested that closure of ASD in patients beyond the age of 60 years is beneficial in terms of improvement of symptoms and functional capacity, reduction of PA pressures and RV dimensions and function and BNP level (Heart 2023;109:1741-1750).



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BALLOON ATRIAL SEPTOSTOMY – BAIL OUT PROCEDURE FOR SEVERE , REFRACTORY PULMONARY ARTERIAL HYPERTENSION



Pulmonary Arterial Hypertension (PAH) is a progressive , heterogenous and highly morbid disease with poor median survival of untreated patients of less than 3yrs in adults and 10 months in children . However , with advancements in early diagnosis and medical therapy targeted specifically against PHT has significantly improved survival and quality of life in the last two decades . Management of PAH generally includes calcium antagonists , anticoagulants , antifailure therapy ,supplement O₂ and specific drugs which includes combination of prostacycline analogs , PD5 inhibitors , Endothelin receptor antagonists and guanyl cyclise inhibitors . Balloon atrial septostomy (BAS) is rarely done for this entity and recommended only in patients with severe pulmonary hypertension and intractable right heart failure despite maximal medical therapy or as a bridge before lung transplantation . Hemodynamic benefits of BAS is mainly due to decompression of RA and RV pressures and due to increase in cardiac output by right – left shut across atrial septum (despite modest reduction in arterial O₂ saturation) improving overall O₂ delivery to peripheral tissues . However , over dilatation of atrial septum may become paradoxically harmful since marked fall in O₂ saturation and severe hypoxia may outweigh the benefits of increased cardiac output .

Balloon Atrial Septostomy (BAS) was done in our patient as a bail out procedure as she was critically ill on life support system despite maximal medical therapy with chances of survival exceedingly low .

FIGURE 1 A



FIGURE 1 B

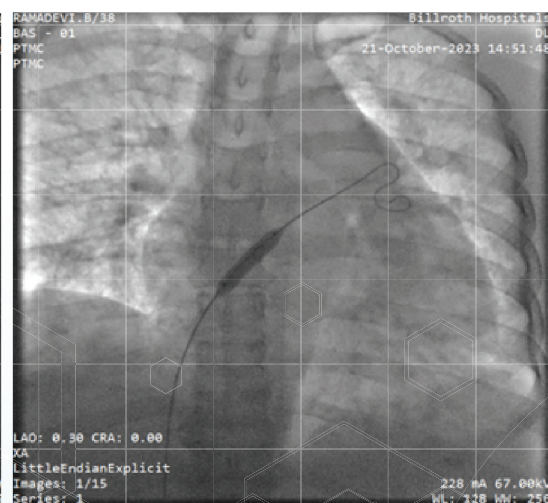


FIGURE 1 A & B SHOWS TRANSEPTAL PUNCTURE AND BALLOON DIALTION OF ATRIAL SEPTUM WITH 6mm and 8mm balloons .



38 year old known case of hypothyroidism and suspected Sjogren 's syndrome on steroids was admitted on 15/10/2023 with c/o dyspnea class III , bilateral pedal edema , abdominal distension , oliguria and palpitation since 3 days . On examination , vital signs include PR – 105/mt , BP120/80,SpO2 94%in room air. ECG showed sinus tachycardia .ECHO showed features of severe PHT – RA , RV dilated , PASP -111mmHg , EF 64%, moderate TR, TAPSE 9mms , IVC dilated and non collapsing , mild pericardial effusion , TAPSE /sPAP ratio 0.08 , ePLAR 0.50 - all features stratifying this patient as high risk category . CT abdomen showed ascites and portal vein thrombosis . Blood investigation showed elevated LFT and renal parameters . She was seen by multidisciplinary team , diagnosed as severe pulmonary arterial hypertension (group 1) with right heart failure and started on anti failure therapy , anticoagulants , steriod , O2 supplement and titrated doses of triple therapy with sildanafil , Bosentan and selexipeg . Ascitic tapping was done on 17/10/2023 and 1.2litres of fluid was aspirated . Despite above therapy , she continued to deteriorate and put on life support system with assisted ventilation and inotrope infusion . At this stage , she was recommended for BAS as bail out procedure since the chance of survival was very poor.

FIGURE 2 A

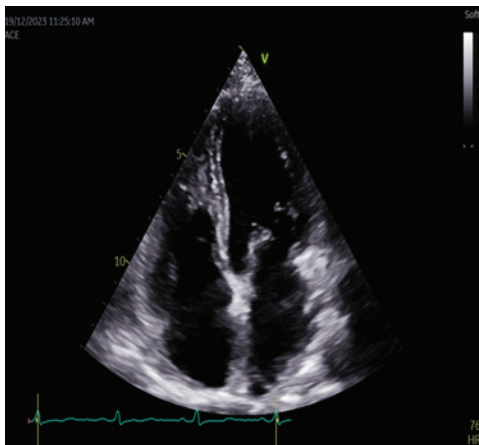


FIGURE 2 B



FIGURE 3C

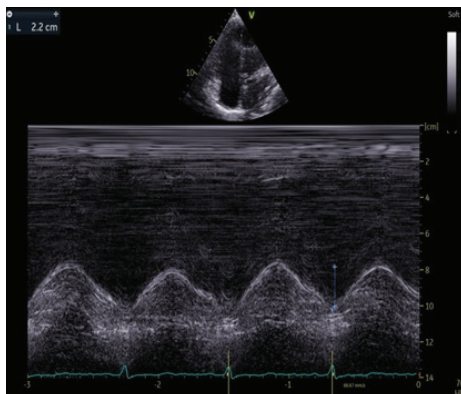


FIGURE 3D

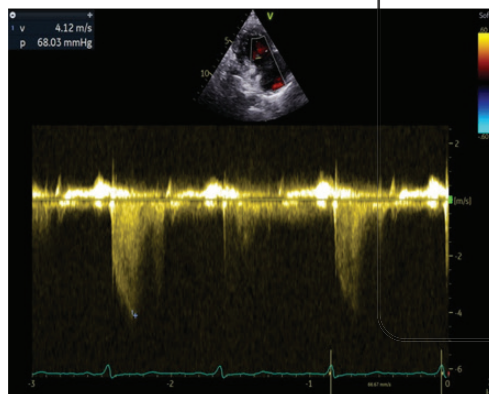


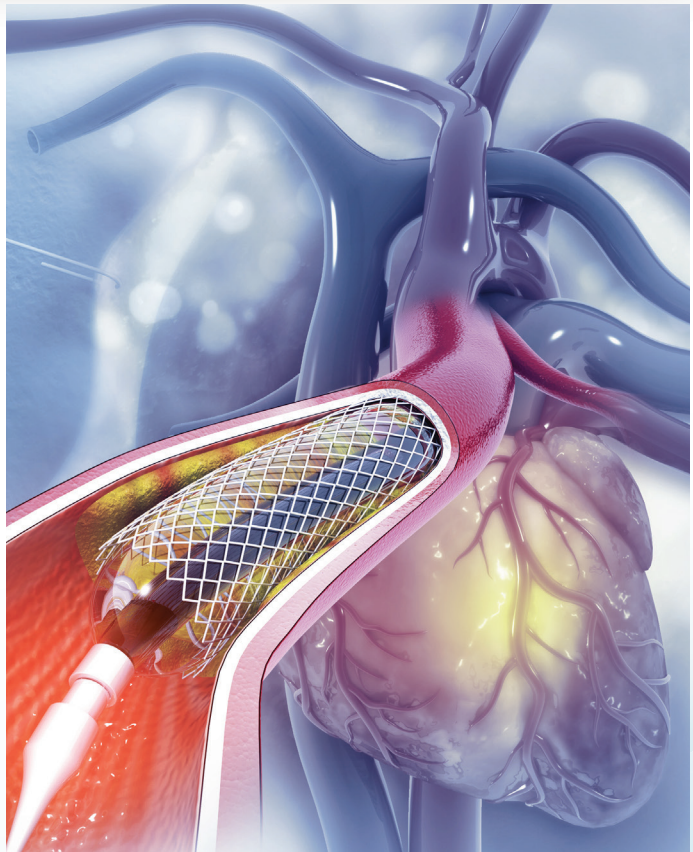
Figure 3A 3B 3C 3D contains follow up echo images of the same patients 5 months after procedure with normal RA RV dimensions, pulmonary artery pressures, normal TAPSE and biventricular function and no pericardial effusion .

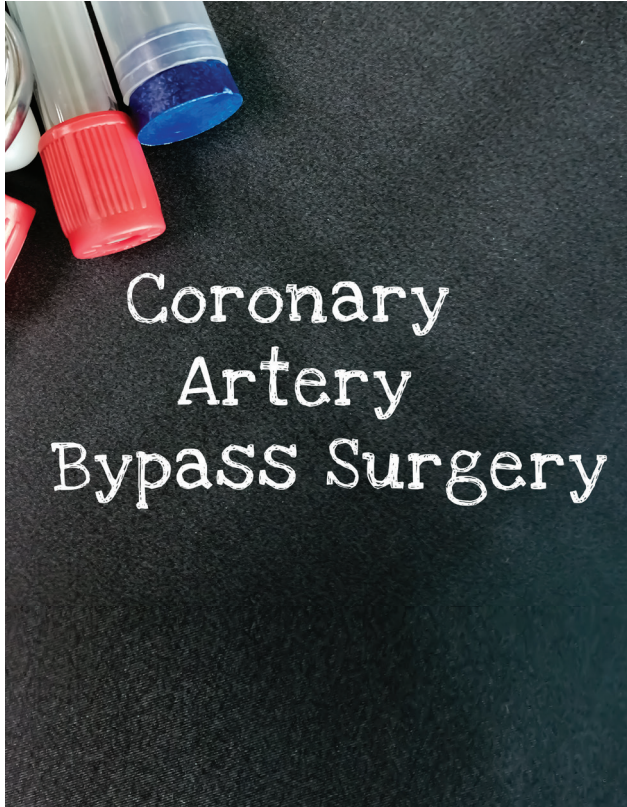
BLOCK THE STORM



Electrical storm or Ventricular storm refers to multiple recurrences of ventricular tachycardia/ventricular fibrillation (VT/VF). It is obviously a life-threatening situation and the prognosis is dismal without proper management. This is often refractory to conventional therapies and require additional procedures. We report herewith our experience with five such patients encountered in the last two years.

CASE 1: MG a 50 year old male presented with ACS – ST elevation MI of inferior wall and RV. He had features of RV dysfunction in echo and RV failure clinically. Angiography revealed left main with triple vessel disease with occluded RCA. The initial plan was to stabilize him and perform CABG. In view of progressive RV failure, delayed PCI to RCA was done which improved his RV failure. 2 days later OP CABG was done and received 2 grafts. On second post operative day he developed frequent VPCs, VT and VF. He required almost 25 DC shocks. Left SGB was done (neurolysis with 60% alcohol) under ultrasound guidance. VT recurred 12 hours later. Right SGB was then done and Ropivacaine was infused for 5 days. There was no recurrence of VT and he is doing well for 3 years now.





CASE 2: M 57 year old male was admitted in September 2023 for ACS (NSTEMI) with LV dysfunction associated with pneumonia. His ECG showed ST T changes suggestive of anterior wall ischemia. Echo showed regional wall motion abnormalities of LAD territory an EF of 37% and grade II MR with pulmonary hypertension and RV dysfunction. Coronary angiography revealed left main with triple vessel disease and he underwent emergency CABG and received 5 grafts. On postoperative day 2 he had VT/VF which was reverted by DC shock (4 times). Next day re required almost 30 shocks to treat recurrent VT. He received Sotalol and Mexiletine and in view of recurrent VT, bilateral stellate ganglion blockade was performed after which there was not even a single arrhythmic event. But he developed vocal cord paralysis followed by aspiration pneumonia and sepsis to which he succumbed.

CASE 3: 68years old male with history of Sinus node dysfunction for which pacemaker was implanted 2 years ago. He presented with Acute heart failure, with severe LV dysfunction (EF-28%). He was intubated and subsequently he had multiple episode of VT for which many electrical cardioversions were done. Left sided stellate ganglion block was done with USG guided with Ropivacaine. After stabilization he underwent Cardiac resynchronization therapy with defibrillator (CRT-D) Implantation.

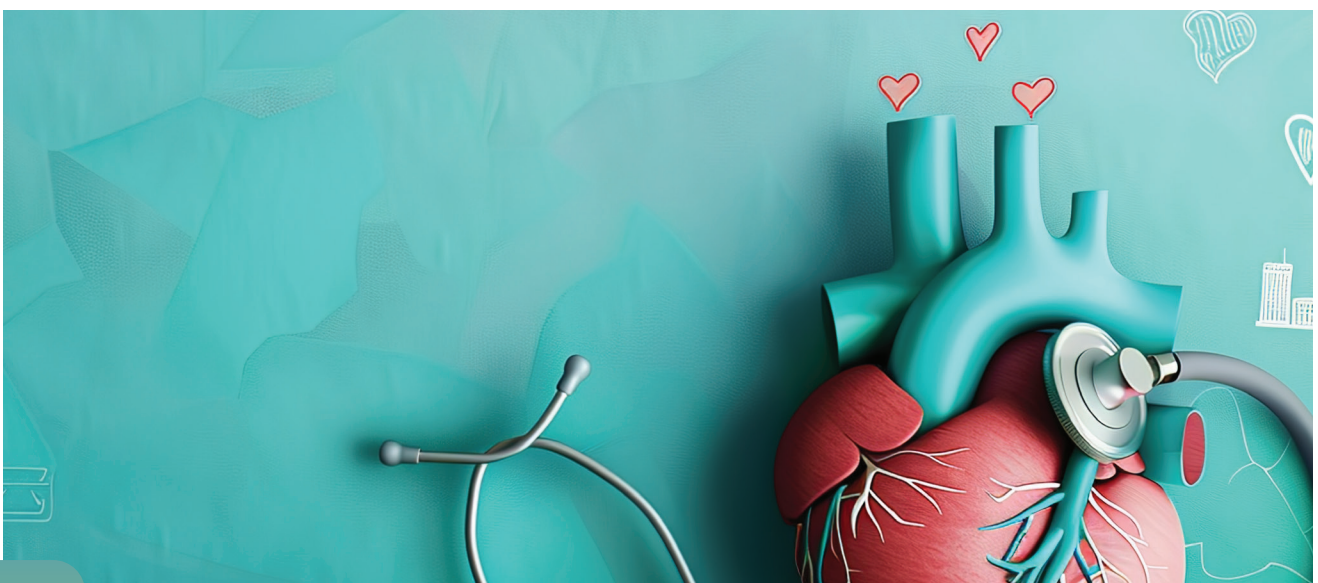
He is on regular fallow up. LV function has improved and there was no recurrence of VT.





CASE 4: 65years old male , known case of Ischemic cardiomyopathy , severe LV dysfunction and post primary PCI to LAD had received an ICD for primary prevention. On admission he manifested recurrent VT for which cardioversion was done and patient was intubated. Bilateral sided stellate ganglion block was done with USG guided with ropivacaine. He developed recurrent VT and died.

CASE 5: 58 year old male had received a single chamber ICD in 2018 for primary prevention following an anterior wall STEMI associated with LV dysfunction. His recanalized LAD did not require PCI. He was doing well except that he received 2 shocks in the last 6 years. Recently he was diagnosed to have adenocarcinoma stomach for which total gastrectomy with gastrojejunostomy was done. On postoperative day 2, he developed repeated VT/VF and he received multiple shocks from ICD. He received intravenous Metoprolol in 6th hourly boluses, Amiodarone infusion and Mexiletine by Nasogastric tube for VT but none helped. Next day also he received multiple ICD shocks mostly for VT/VF (appropriate) and some for atrial fibrillation (inappropriate). He was intubated and mechanically ventilated with intravenous sedation and muscle relaxant. Left stellate ganglion blockade was done at the bedside with USG guidance and Ropivacaine was infused for 2 days. There was no recurrence of VT but atrial fibrillation recurred and in order to avoid inappropriate shocks reprogramming was done by changing VT recognition zone. Since the ICD was almost totally depleted, replacement of the ICD device with CRT-D was planned but he developed sepsis due to anastomosis leak in abdomen. He was discharged at request.





DISCUSSION:

The term electrical storm includes the occurrence of three or more episodes of sustained ventricular arrhythmias within 24 hours or VT recurring within 5 minutes of termination of another episode or sustained or non-sustained VT with the total number of ventricular beats exceeding sinus beats in 24 hours. This may occur in those with underlying structural heart diseases (SHD) like post myocardial infarction state, cardiomyopathy, aortic valve disease etc. and may be documented by device interrogation of patients with Implantable Cardioverter Defibrillator (ICD). It may also occur in those without SHD as in Brugada syndrome, Long QT syndromes etc. Roughly 4-7% of patients who received ICD for primary prevention and 10-30% of those who received ICD for secondary prevention may experience this condition. The usual triggers are drug toxicity, electrolyte disturbances, new or worsened heart failure, acute myocardial ischemia, thyrotoxicosis and QT prolongation. The presentation may be in the form of palpitations with presyncope or syncope or may present as cardiac arrest. Diagnosis is done by Holter ECG monitoring or by interrogation of the device. Because of the life-threatening potential of electrical storm, it is better to hospitalize and monitor them with telemetry. Treatment is in the form of Electrical cardioversion if the patient's hemodynamics is unstable. Amiodarone with betablocker (iv esmolol or metoprolol) is usually needed for acute as well as long term management. Propranolol, a non-selective beta blocker, is more effective than selective beta blockers. Quinidine may work at times. Mexiletine may be considered if other antiarrhythmics are ineffective. Revascularization is indicated, particularly when VT is polymorphic. Electrolyte abnormalities like hypokalemia, hypomagnesemia and hypocalcemia should be corrected. If potassium is administered the blood levels should be increased to the upper limit of normal range. Magnesium may help even if serum level is normal. In refractory cases, Deep sedation, General or Epidural anesthesia, insertion of IABP or any other mechanical circulatory support, Left or Bilateral Stellate ganglion blockade (SGB) or stereotactic body radiation therapy may be considered. Renal artery denervation has been found to be useful in a small series of patients. Surgical Cardiac sympathetic denervation may be needed if SGB fails or is difficult to perform. Radiofrequency ablation of VT may be helpful though recurrences are likely. Cardiac transplantation may be considered, if refractory to all these measures.



SGB PROCEDURE:

Left stellate ganglion blockade is performed first because it is most often effective, requires shorter time and is more suitable in conscious or emergency patients. Bilateral SGB is only performed in patients who are under general anesthesia not responding to a unilateral block. In intubated patients, if a unilateral block is unsuccessful within 10 minutes, consideration would be made for a bilateral block. In patients who are not intubated, bilateral SGB is not considered because of risk of phrenic nerve blockade and respiratory distress. The occurrence of SGB was evaluated by assessing the increase in temperature in the ipsilateral arm.

ULTRASONOGRAPHY-GUIDED SGB

All pertinent structures are identified by ultrasonography (carotid artery, left internal jugular vein, longus coli muscle, vertebral artery, anterior scalene muscle, and brachial plexus). The skin is anesthetized with 2 mL of 1% lidocaine. A 22-gauge, 2-inch spinal needle was advanced in-plane in a posterior-to-anterior direction to the anterior surface of the longus coli muscle to avoid all vascular structures. After a negative aspiration, local anesthetic agents were injected incrementally. The stellate ganglion is visualized lifting off the anterior aspect of the longus coli muscle

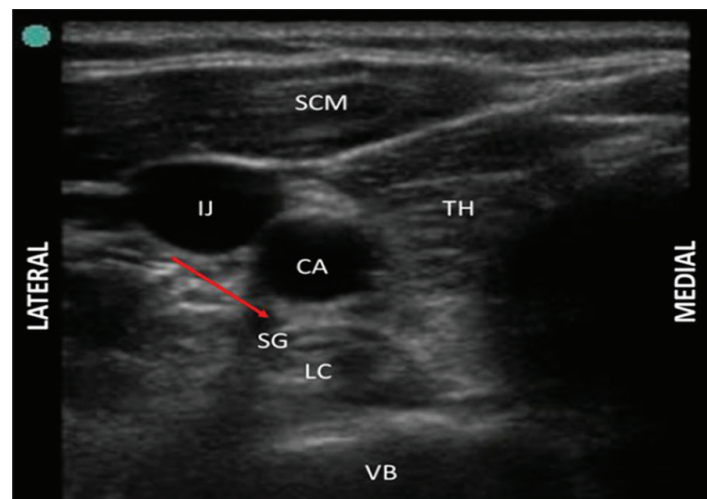
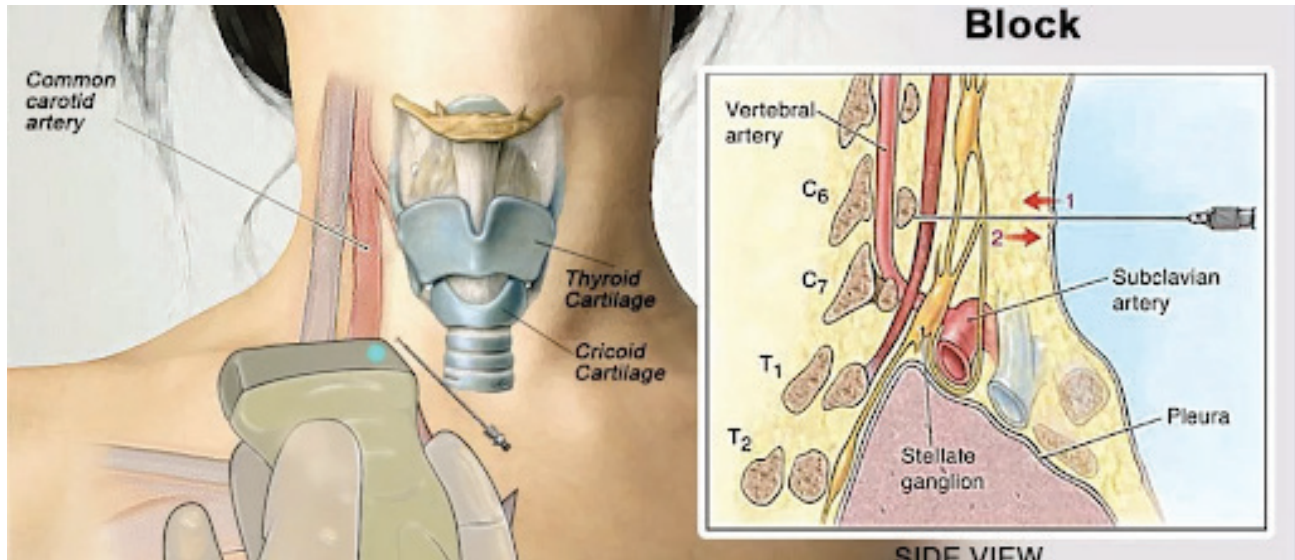


Image of stellate ganglion blockade guided by ultrasonography. The red arrow shows the path of the needle. CA indicates carotid artery; IJ, internal jugular vein; LC, longus colli muscle; SCM, sternocleidomastoid; SG, area of stellate ganglion; TH, thyroid; and VB, vertebral.



FLUOROSCOPY-GUIDED SGB

The anterolateral points of entry on the skin, targeting the uncinate processes of C6 bilaterally, are infiltrated using 1% lidocaine. Care is taken to avoid puncturing any preexisting appliances in the neck. Under fluoroscopic guidance, a 3.5-inch, 25-gauge spinal needle is advanced to gently contact the uncinate process/base of the transverse process. After a negative aspiration, 0.5 mL of contrast media was injected, which had to show a nonvascular spread pattern in the region of the stellate ganglion. Next, local anesthetic agents are slowly and incrementally injected after frequent negative aspirations.

0.2% Ropivacaine is commonly used and administered as infusion at a rate of 5 mL/hour using a catheter kept in place. Alcohol injection results in neurolysis and bilateral alcohol injection is discouraged. Contraindications to performing a stellate ganglion nerve block include recent history of a myocardial infarction, anticoagulated patients, coagulopathy, glaucoma, and patients with a preexisting cardiac conduction blockade.

MESSAGE:

SGB is effective in > 50% of cases of electrical storm. It is relatively a simple and safe procedure in the experienced hands. It can be done in ICU with USG guidance or in the cathlab by fluoroscopic guidance. Complications such as accidental intravascular injection, brachial plexus and recurrent laryngeal nerve palsy, injury to trachea, and esophagus are known but fortunately rare. Left SGB can also result in an imbalance in myocardial contractility and asynchrony of the left ventricle. Hence, there is an attached risk of residual left ventricular dysfunction.

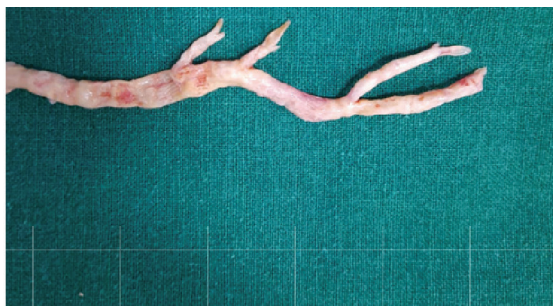
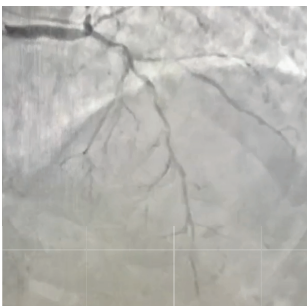
CASE SERIES OF CORONARY ENDARTERECTOMY A SINGLE CENTRE EXPERIENCE



INTRODUCTION

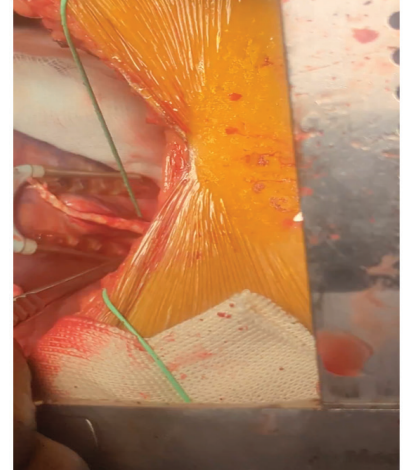
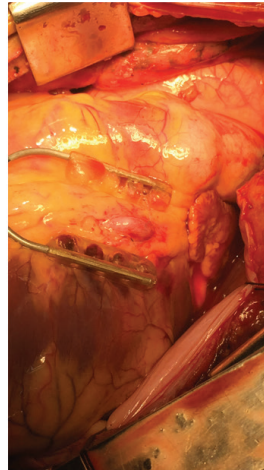
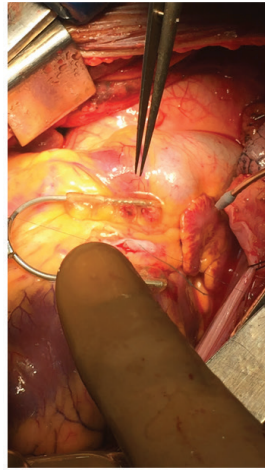
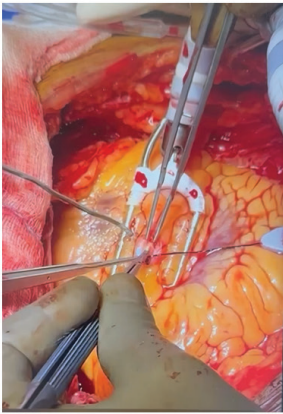
In patients with diffuse coronary artery disease (CAD) undergoing coronary artery bypass grafting (CABG), severe calcification may render the creation of distal anastomoses challenging or impossible, leaving regions of viable myocardium at risk for ischemia. In Recent times compelling requirement complex CABG, due to diffuse CAD and diabetes mellitus, renal insufficiency, peripheral vascular disease and previous PTCA for 1-2 VD which often requires endarterectomy.

Coronary endarterectomy (CE) is a rare technique used with CABG (CE-CABG), typically either to facilitate the revascularization of otherwise ungraftable targets or as an unplanned bailout after opening of a severely calcified artery where the creation of a distal anastomosis would be otherwise impossible. In this case series, we analysed patient underwent coronary endarterectomy at our center.



RESULTS

We retrospectively analysed our Hospital's cases of CABG done between Jan 2022 to Dec 2023. In year 2022 21 patients underwent endarterectomy of total 301 CABG. In year 2023, 162 endarterectomies out of 481 CABG were done. Total number of endarterectomy $n=183$ (23%). Of these 7 patients underwent on pump endarterectomy and remaining are off pump endarterectomy ($n=176$). Of 183 endarterectomies, 162 patients had single vessel, 20 patient had two vessel and 1 patient had single vessel endarterectomies. Our early results were satisfactory, where 3 patient revealed significant ST changes in ECG, 5 patient had anticoagulation related significant bleeding postoperatively and during followup, none of the patient early post op mortality, one patient had late post-operative mortality. 3 patients check angiograms were done during followup, were patent grafts and good runoff noted in all three patients.



CONCLUSIONS

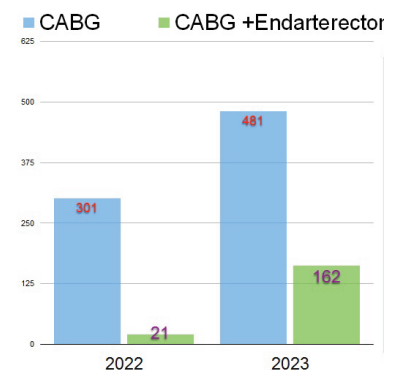
This analysis demonstrates that CE-CABG has acceptable long-term outcomes and serves as a benchmark for what can be expected when this rare procedure is used. CE is a rarely used technique to approach difficult targets in coronary surgery in past,

This analysis of a large cohort of patients in the STS ACSD demonstrates that CE-CABG has acceptable outcomes with mildly increased rates of early mortality and postoperative MI but similar long-term survival compared with CABG alone.

Our findings suggest that the risk associated with adjunct CE is not prohibitive for patients requiring advanced revascularization techniques for severe CAD. However, the decision to perform CE-CABG should not be taken lightly. Surgeons should carefully consider which patients may benefit from CE-CABG as our data suggest that the technique is associated with increased risk that should be justified by potential benefit.

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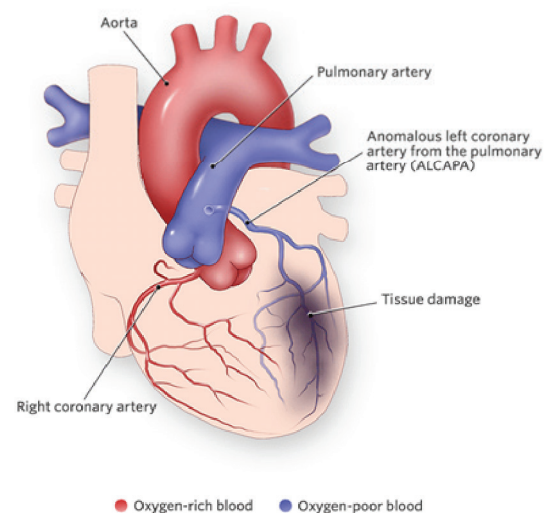
A RARE CHD: ANOMALOUS LEFT CORONARY ARTERY FROM PULMONARY ARTERY (ALCAPA) PRESENTING IN ADULthood COMPLETE SURGICAL CORRECTION (DUAL CORONARY SYSTEM)



INTRODUCTION

Anomalous left coronary artery from pulmonary artery (ALCAPA) is a rare congenital heart disease that affects one in every 300 000 live births and accounts for 0.24–0.46% of cases of congenital heart disease. Over the last few decades, four different operative procedures have been recommended as treatment for ALCAPA: the simple ligation of ALCAPA, coronary artery bypass grafting, channel repair (Takeuchi surgery), and coronary artery reimplantation. The reimplantation surgery of ALCAPA is the current ideal option to achieve a definitive two-coronary artery anatomy and physiology, and has a good short-term and long-term outcome; it has become the first-choice procedure for this anomaly. We report a new modified technique to repair ALCAPA in an adult.

Anomalous Left Coronary Artery from the Pulmonary Artery (ALCAPA)



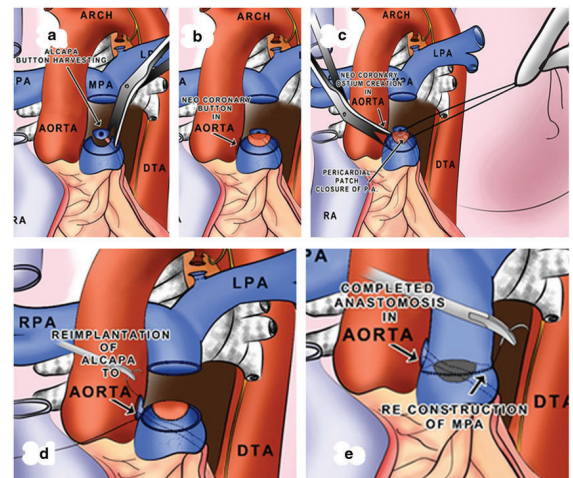
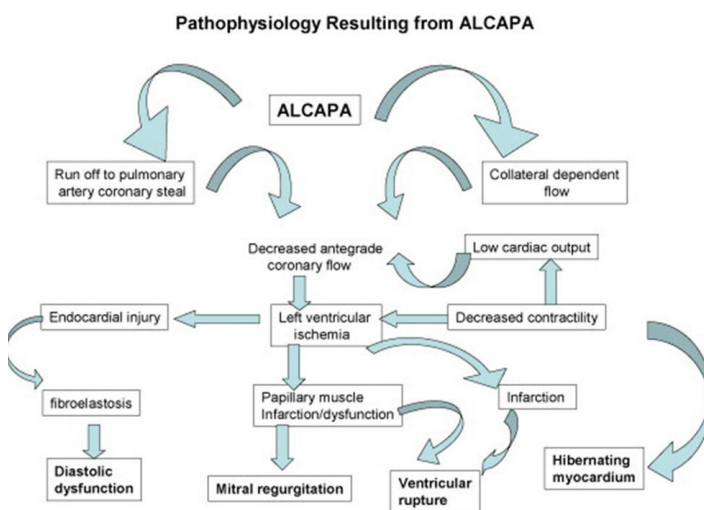
CASE REPORT

Mr.VK, 26Years /male, Police by occupation, actively doing Gym exercise, on evaluation elsewhere for emergency appendicectomy (Dec 2023), patient underwent cardiac evaluation in view of ECG changes. His ECHO revealed grossly dilated RCA -7mm in size. LCA origin from aortic root could not be visualized. Anomalous left coronary artery from Pulmonary artery noted, LCA across from the PA - dilated, 8mm in size. There was continuous flow inside the PA from LCA. Multiple muscular VSD with left to right shunt -likely to be collaterals. No regional wall motion abnormality. Normal LVEF 58%). Mild MR. No PAH. CT coronary angiogram was suggestive of ALCAPA.

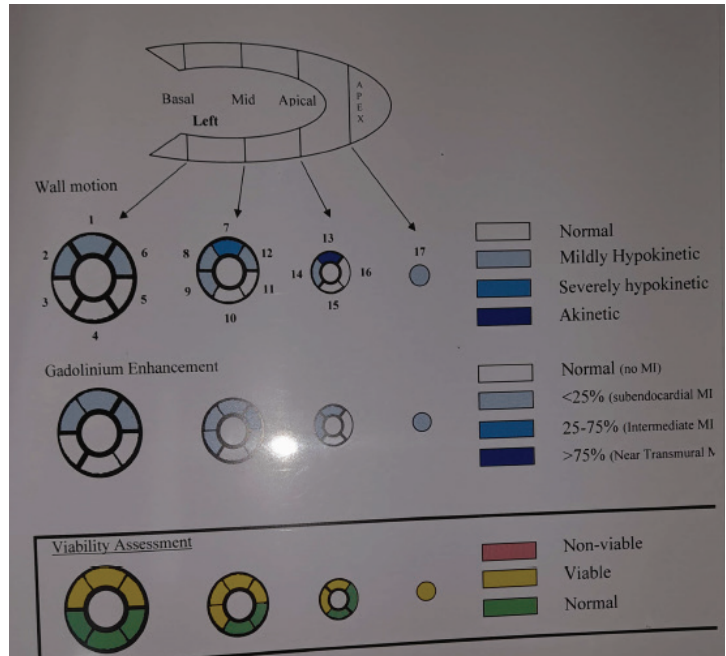
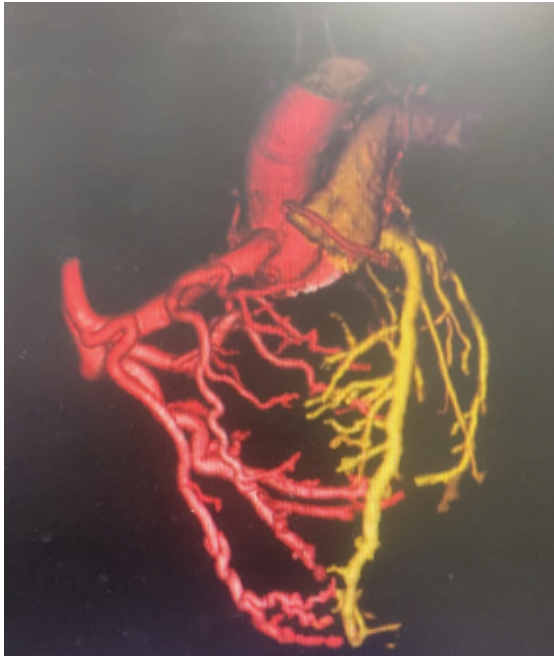
Patient came to our hospital for further management. Presented with NYHA Class I-II dyspnea on exertion/atypical chest pain. C-MRI was which done revealed dilated LV with EF45%, concentric LVH, subendocardial infarcts - anterior & septal wall, viable myocardium 10/17 segments. Conventional angiogram revealed - dilated RCA, LCA filling through inter-coronary collaterals which was dilated and tortuous draining into MPA.



On 30-03-2024, he underwent ALCAPA repair- by Left Coronary artery transfer to Aorta -Dual coronary system technique. Routine CPB established, Aorta - PA groove dissected, Aorta - PA Looped,. Patient cooled gradually to 24 degrees. Tiny collaterals across PA- Aorta were ligated. Aorta cross clamped. Antegrade Del Nido cardioplegia given through aortic root. Main Pulmonary artery transected at mid level. After confirming the findings, it was decided to do direct re-implantation of left main coronary artery. Aorta was transected at level of sino-tubular junction. Left coronary button harvested. Surrounding tiny collaterals ligated. RPA mobilized from posterior wall of aorta. Left coronary button reimplanted over the posterior wall of aorta of STJ parallel to level of RCA ostia using 5-0 prolene sutures. Reimplanted button on aorta checked for leak. Transected aorta closed in layers using 4-0 prolene. Deficient posterior wall - PA (LCA Button) was closed using gluteraldehyde treated pericardial patch. Transected MPA was sutured using 4-0 prolene suture. Patient rewarmed. Tissel hemostatic agent applied over arteriotomy and anastomosis sites. Routine weaning and closure done.



He was shifted to ICU with stable hemodynamics and was extubated after 3 hours of shifting to ICU. His drains were removed on 1st POD. On POD-3, Patient developed hypotension (Bp-90/60mmhg) and severe profuse sweating, Echo and X ray showed large pericardial effusion. Pericardiocentesis was done via subxiphoid incision and he was shifted to ICU with stable hemodynamics. He was extubated on the same day. He was mobilized well and made good recovery. His sutures/ pacing wire were removed and discharged on 9th post-operative day. Currently he is in regular followup, clinically asymptomatic and back to his routine Job.



CONCLUSION

Dual-coronary system can be established in patients with ALCAPA. Coronary extension implantation techniques have acceptable operative mortality and excellent cardiac recovery and late survival. Although the rate of late coronary occlusion is low, continual ventricular or mitral valve dysfunction should trigger evaluation of persistent coronary compromise.

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VSR - A CASE REPORT



INTRODUCTION

Postinfarction ventricular septal rupture (VSR) remains an important and life-threatening complication of myocardial infarction (MI). Although several techniques have improved surgical outcomes of post-infarction VSR, the treatment is still challenging with a high mortality rate. In our center we do follow infarct exclusion technique.

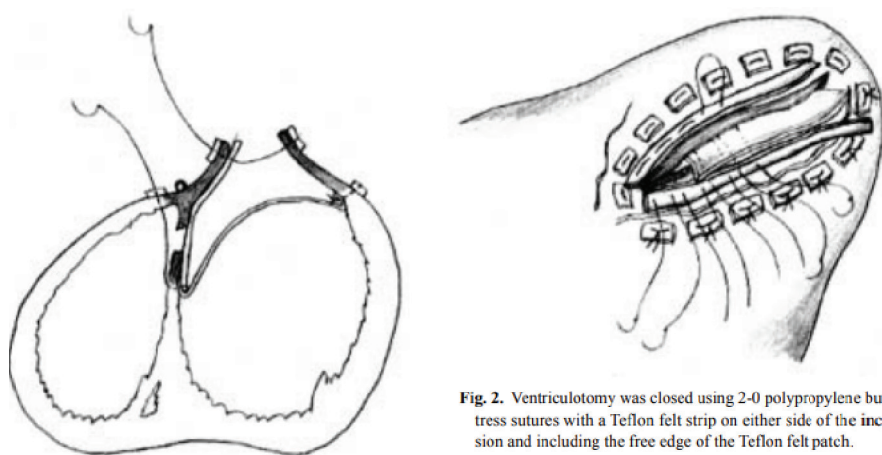


Fig. 2. Ventriculotomy was closed using 2-0 polypropylene buttress sutures with a Teflon felt strip on either side of the incision and including the free edge of the Teflon felt patch.

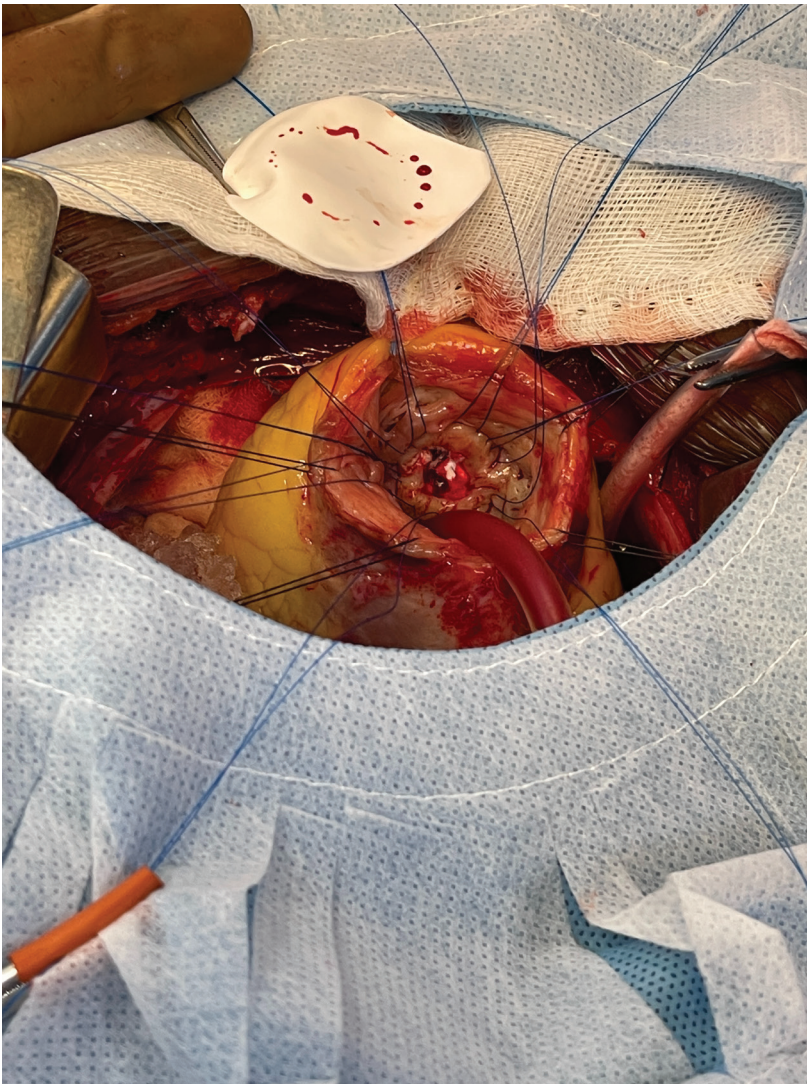
CASE REPORT

Mr.G, 78yrs/male, known diabetic, hypertensive for past 10yrs on regular medications, presented initially elsewhere with complaints of chest pain and breathlessness for 1 week duration. His ECG was suggestive of AWTMI (ST-Changes in VI-V5 leads). ECHO revealed ventricular septal rupture across apical septum measuring 15mm with left to right shunt (PG: 68mmhg).severe tricuspid regurgitation (TRPG:60mmhg). RWMA+. Mid Anteroseptum, Apical Septum, Anterior, Inferior, Lateral Segments Are Thinned And Severely Hypokinetic / Lv Apex Dyskinetic.Mild Left Ventricular Systolic Dysfunction(LVEF: 47%) Grade II LV Diastolic Dysfunction. Mitral Regurgitation (Mild).

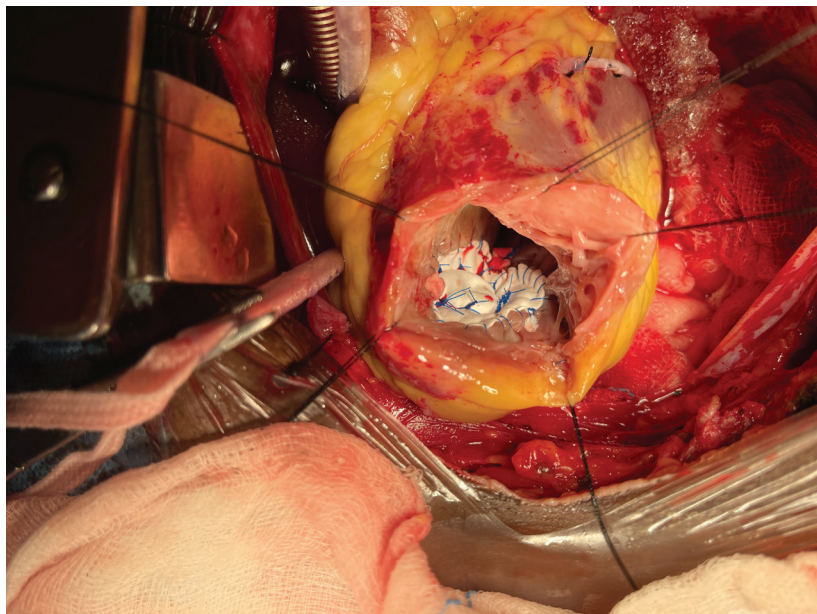




He underwent VSR- Apical type (Infarct Exclusion) Bovine Pericardial Patch closure + Tricuspid Valve Repair with 3D contour Medtronic ring repair on 08.07.2024. Routine CPB & diastolic heart arrest, LV opened near apex parallel to LAD 22cm x 3 cm. Apical ventricular septal rupture noted with surrounding inflamed septal myocardium. Ventricular septal rupture was repaired by septal infarct exclusion technique using Bovine pericardium with multiple interrupted and continuous 4-0 prolene pledgetted sutures. Left ventriculotomy closed with 4-0 prolene, reinforced with Teflon felt on both sides. RA opened parallel to RV groove. Dilated tricuspid annulus with severe TR noted. Tricuspid ring annuloplasty done with 28mm contour 3D ring. Tricuspid valve found competent on saline test. RA closed. Tense PA requiring prolonged CPB and IABP support for weaning. He was shifted to ICU with ventilator, pacing, moderate inotropic and IABP supports with borderline hemodynamic status.



Post operatively patient gradually improved with hemodynamic status. He was treated with moderate inotrope supports, pulmonary vasodilators, antitocoagulants, antiplatelets, antiarrhythmics, diuretics infusion and appropriate metabolic corrections. On POD-2 he was taken for re exploration in view of increased chest drain and drop in hemoglobin.



He was continued with all ventilator/inotrope/IABP supports and developed subcutaneous emphysema on right side, additional right side ICD inserted. He had elevated Total count, low Hb 5.6gm%, low platelets (upto 65000), elevated PT/INR/aPPT, Renal and LFT parameters and low sr. Albumin (1.8mg/dl). He was given multiple albumin, blood and blood product transfusion (PCV-10, FFP-10, Platelets-10). His ECHO revealed no residual VSD, moderate RV dysfunction/LV

dysfunction/Trace pericardial effusion. Due to unaffordability, patient was taken to GH for further management on POD-4th, where he was extubated on POD-7th.

CONCLUSION

The repair is simple and durable and has reduced the incidence of residual ventricular septal defects and patch dehiscence.

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