LILAVATI HOSPITAL MEDICAL TIMES

SEPTEMBER 2021-



Contents

CHIEF EDITOR
Dr. Abhay Bhave
,
EDITORIAL TEAM
Dr. Amey Medhekar
Dr. Bhavesh Vajifdar
Dr. Chandralekha Tampi
Dr. D.R.Kulkarni
Dr. Kiran Coelho
Dr. Leena Jain
Dr. Parag Dhumane
Dr. Rajeev Redkar
Dr. Salil Mehta
Dr. Sheikh Minhaj Ahmed
CO-ORDINATOR
Mr. Kundan Singh
All the correspondence should be
All the correspondence should be addressed:
addressed:
-
addressed: To, The Chief Editor
addressed: To,
addressed: To, The Chief Editor Lilavati Hospital Medical Times
To, The Chief Editor Lilavati Hospital Medical Times Lilavati Hospital & Research Centre
To, The Chief Editor Lilavati Hospital Medical Times Lilavati Hospital & Research Centre A-791, Bandra Reclamation, Bandra (W)
To, The Chief Editor Lilavati Hospital Medical Times Lilavati Hospital & Research Centre A-791, Bandra Reclamation, Bandra (W) Mumbai - 400 050.

CHAIRPERSON - LHMT

From COO's Desk
Editorial
Overview: Lilavati Hospital and Research Centre 4
Case Reports
 Cardiac Anesthesia, Cardiology & Cardiac Surgery Anesthesiology Chest Medicine
• Orthopaedics
Paediatric Surgery
Plastic Surgery
List of Publications
Straight from the Heart - Patient Testimonials 29
Services Available
Important Telephone Numbers32
Few Honorable Mentions
Doctors Associated with Lilavati Hospital 35

The views expressed in the Medical Times are not of Lilavati Hospital or the editor or publisher. No part of the Medical Times can be reproduced in any form including printing or electronic without the written permission of the chief editor or publisher. The information provided on medicines, materials, investigations, procedures, therapies and anything medical is the sole responsibility of the author of the article and the hospital shall not be responsible for any such information.



From COO's Desk



We are pleased to present yet another enticing edition of our quarterly magazine - Lilavati Hospital Medical Times (LHMT).

As we move from second phase to a 'threatening' possible third phase, we hope that the pandemic will be under control and we do not have to face the onslaught of the third wave. Hospital though is mentally & physically prepared to deal with this; especially as our experts are voicing a pan India increase in pediatric cases. Throughout this pandemic, the doctors, nurses, other medical and paramedical staff of Lilavati Hospital and Research Centre have proved that in the time of crisis

they will rise to the occasion.

The non covid work has increased, especially the surgical work. Cath lab, OPD, Operation theaters and diagnostic services are swamped with non-Covid patients, and credit goes to each and every employee of Lilavati Hospital. This increasing flow of patients is due to diligent implementation of green, orange and red zones by all the staff of the hospital with absolute segregation of Covid areas.

I would like to extend warm greetings and heartfelt gratitude to all the staff of LHRC for the exemplary services during the second wave of Covid. High end surgeries including Liver transplantation surgeries are being performed as frequently as in pre-Covid times.

I am hopeful for a return to our "new normal". While we are turning the corner, there may be a few bumps along the way.

Our vaccination programme is going on full throttle. We will continue to improve our facilities and services, bring the newest in medical technology to our community, and keep our team committed to the "Patient First" and "More than healthcare, human care" ethic.

We are thankful to the readers for their overwhelming response for previous editions. We are publishing interesting case reports and studies from the therapeutic & diagnostic sides in this edition. The Cardiac, Respiratory medicine (adult & pediatric), Orthopedic surgery, Plastic surgery and Pediatric surgery teams have taken painstaking efforts to bring this content to you.

Best wishes and Greetings to all for the ensuing festive season.

Lt. Gen. (Dr.) V. Ravishankar

Chief Operating Officer and Consultant Cardiothoracic Surgeon

Editorial



At last, we can begin to breathe easy with the ebbing covid onslaught. The entire hospital efforts have resulted in less morbidity and mortality in this wave of covid, and here's hoping the next wave if any would be gentle on us. I am sure our hospital team members have learnt so much that they can effectively manage any new onslaught. We salute all these warriors and thank them profusely!

The hospital OPD attendance has increased and the hospital admissions and departments are almost back to the pre covid era in terms of their functioning. Importantly the vaccination program in the hospital is going at full speed and has

brought back patients to the hospital – a welcome trend of course.

Similarly, I am glad that covid did not dampen our spirit to publish in the Lilavati Hospital Medical Times with interesting case reports and events published in this foray for which I once again thank the COO Dr. V Ravishankar, office bearers of the publication and the medical community in the hospital for their full cooperation, to the extent that with the help of marketing we were able to do an extra publication (for the first time) on the World Plastic surgical day that was a roaring success! We welcome more departments to use this initiative.

While we are getting back to a normal life, we should continue to be wary of and prevent Covid spread ,by awareness amongst each one of us to maintain distance, clean hands, use sanitizer and avoid crowding. We continue to appeal to our patients and the general community to get vaccinated so as to reduce the risk of infection.

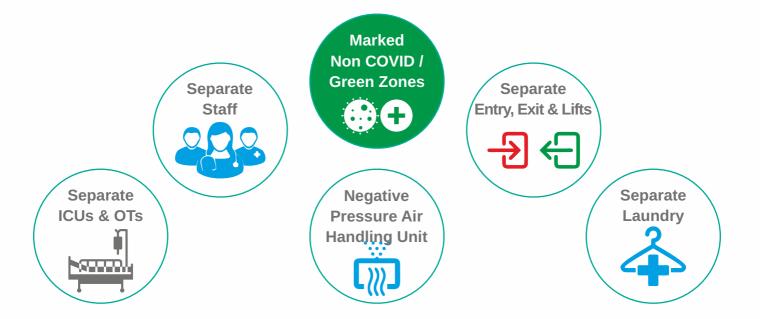
Our list of publications from the hospital staff in national and international journals has continued to be impressive and the research work has gone uninterrupted as have our training programs for the post graduates. Please do read the articles in this publication that highlight the body of fabulous work done in the institution by doctors from different specialties indicating that work went on as usual.

Once again, I request you to read and enjoy this magazine cover to cover to maximally utilize the presented information and hope we are able to impress you with the kind of cutting edge work that is going on in our hospital and the positive impact it has on patient care

Do continue to give us a feedback with criticism and/ or suggestions to assist us in improving the publication- after all we are here to empower you with more knowledge, the more we share the more we learn!

Dr. Abhay A Bhave MD, FRCPA, Haematologist

OUR NEW NORMAL IS AS SAFE AS EVER





PANDEMIC IS TEMPORARY

OUR DEDICATION TO PATIENT CARE AND SAFETY IS PERMANENT

Overview: Lilavati Hospital & Research Centre



Late Shri Kirtilal Mehta



Late Smt. Lilavati K. Mehta

Lilavati Kirtilal Mehta Medical Trust

Lilavati Hospital and Research Centre is run and managed by Public Charitable Trust - Lilavati Kirtilal Mehta Medical Trust which was formed in 1978. The Trust was started by late Shri Kirtilal Manilal Mehta. The Trust has engaged in innumerable charitable endeavors across India.

The Lilavati Kirtilal Mehta Medical Trust is being managed and administered by Board of Trustees:			
Shri Nanik Rupani			
Shri Rashmi K. Mehta			
Shri Dilip Shanghvi			
Shri Chetan P. Mehta			
Shri Bhavin R. Mehta			
Shri Ayushman C. Mehta			
Principal Advisor to the Board of Trustees and Lilavati Hospital & Research Centre Shri S. Lakshminarayanan, IAS (Rtd.)			

Lilavati Hospital And Research Centre

Late Shri Vijay Mehta wished to fulfill his parents desire to build a world-class hospital where everyone in need for relief from disease and suffering come in with a certainty to receive the best possible medical care. His passion, attention to details and perseverance resulted in iconic healthcare landmark called **Lilavati Hospital**.

Lilavati Hospital & Research Centre is a premier multispecialty tertiary care hospital located in the heart of Mumbai, close to the domestic and the international airport. It encompasses modern healthcare facilities and state of art technology dedicatedly supported by committed staff.

Lilavati Hospital has focused its operation on providing quality care with a human touch; which truly reflects the essence of its motto, "More than Healthcare, Human Care". Being a centre of medical excellence where technology meets international norms and standard, the hospital has got what it takes to be a pioneering quality healthcare institute that is also one of the most sought after and patient friendly hospital.

Mission: To provide affordable healthcare of international standard with human care

Motto: More than Healthcare, Human Care



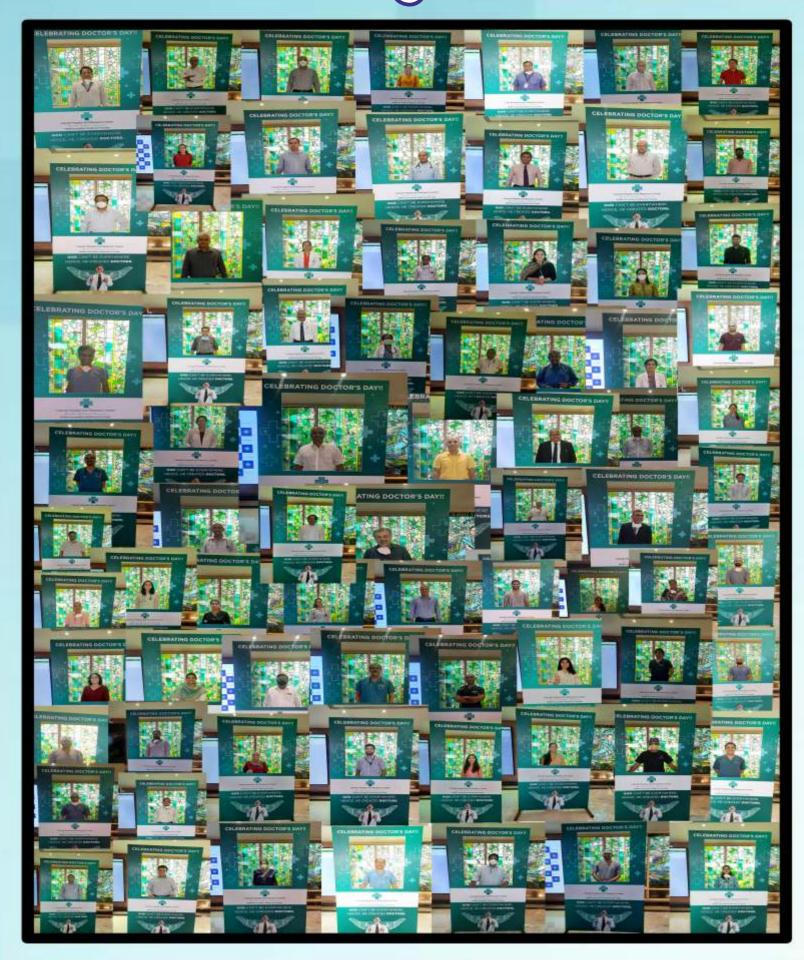
Highlights

- 323 bedded hospital including 77 intensive care beds. Currently number of beds have been temporarily increased for helping fight the COVID pandemic. We have dedicated 144 ward beds and 48 ICU beds to treat COVID positive patients.
- 12 state-of-the-art well equipped operation theatres.
- Full-fledged Liver Transplant, Heart Failure, Hypertension, Bariatric, Foot and Ankle, Dental and Dermo Cosmetology Clinic.
- State of art PET SPECT CT department.
- Lilavati Hospital is equipped with Coronary GRAFT Patency Flowmeter which is first of its kind in India. This imaging system is used in Cardiac surgery to assess GRAFT flow / perfusion in coronary bypass surgery.
- The hospital has installed state-of-art Philips Azurion 7F20 in its cath lab. This is the first of its kind high end
 configuration system installed in India. The new system enables excellent imaging for Coronary, Cerebro &
 Peripheral Vascular Diseases.
- The department of Invasive Cardiology has been upgraded with the addition of a High Definition Optis Mobile OCT (Optical Coherence Tomography) system. It has the latest configuration which gives better 3 Dimensional perspective of Coronary Artery before and after stent deployment.
- The hospital has added Intraoperative Nerve Monitoring system which enables surgeons to identify, confirm and monitor motor nerve function of the patients which helps to reduce the risk of nerve damage during various operative surgeries.
- The hospital has upgraded its ENT department by adding a top-of-the line surgical operating microscope to carry out various microsurgeries under high magnification. The microscope electronics allows the surgeon to electronically control object focusing, magnification, illumination, surgical recording, etc.
- All days round the clock OPD Pathology and Radiology investigations without any Emergency charges.
- ICU Emergency charges after 8pm are kept at par with the day time and additional charges are withdrawn.
- More than 300 consultants and manpower of nearly 1,800.
- Hospital attends to more than 10000 In-patient, 40000 Out-patient and performs thousands of surgeries every year.
- Modern Cathlabs having specialized SICU & ICCU with highly trained cardiac care medical staff.

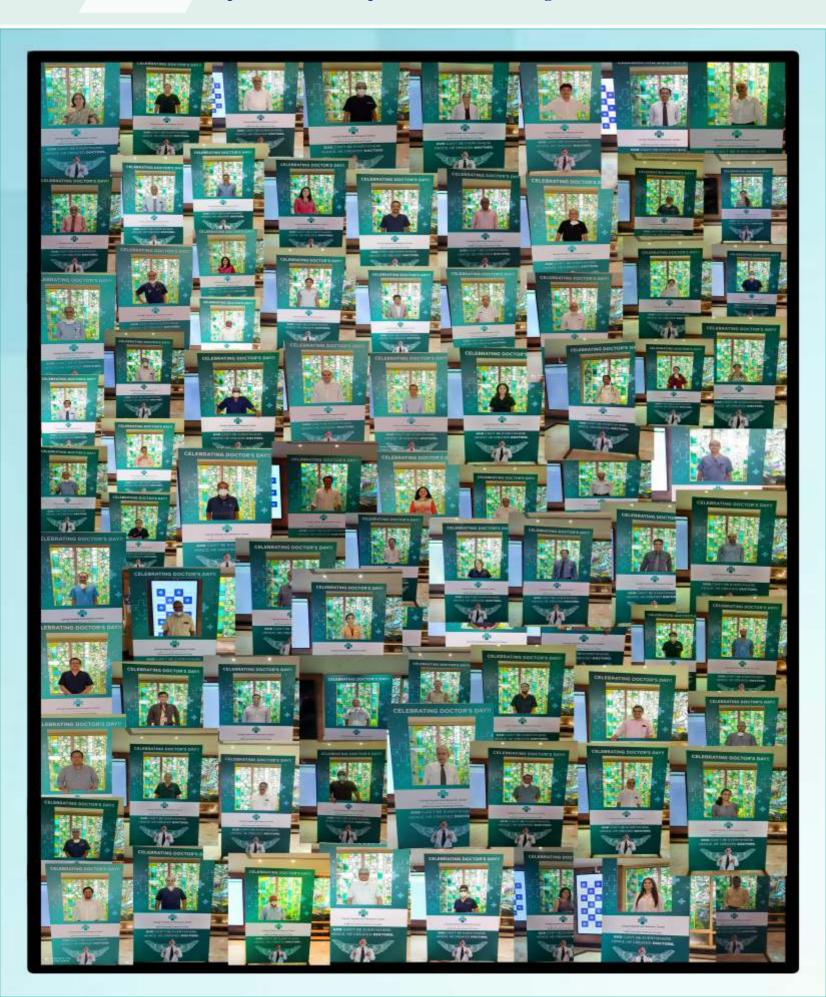
Lilavati Kirtilal Mehta Medical Trust Research Centre

The Lilavati Kirtilal Mehta Medical Trust Research Centre is a Scientific and Industrial Research Organization approved by Ministry of Science and Technology (Govt. of India). The Research Centre under guidelines of Dept. of Science & Technology works in close collaboration in evaluating and developing technologies for better healthcare to the sick people. The research centre has undertaken multidisciplinary researches in the fields of Cardiology, Radiology, Cerebrovascular Diseases (Stroke), Ophthalmology, Chest Medicine, Nuclear Medicine, Pathology, Oncology, Orthopedics etc., to cite a few. One of the important aim of the research centre is to establish community based epidemiological researches in cerebrovascular disease in stroke. As a policy, Drug and Device Trials are not undertaken at the Research Centre.

Doctor's Day Celebration



Watch our esteemed doctors express their love and respect for Lilavati Hospital with some impressive, heart touching, and warm words



CASE REPORT I: Cardiac Anesthesia, Cardiology & Cardiac Surgery

Mitral paravalvular leak closure using AVP III device percutaneously.

Dr. Namrata Kothari, MD, DA (Anesthesiology)

Dr. Ravindra Singh Rao, MD, DM, FACC

Dr. Vidya Suratkal, MD, DM, FACC

Dr. G. N. Rachmale, MS, Mch, MBA

Dr. Ramesh Dargad, MD (Cardiology)

Lt. Gen.(Dr.) V. Ravishankar, VSM, Chief Operating Officer (Lilavati Hospital), MS (General Surgery), DNB (General Surgery), MCh. (Cardiothoracic Surgery), Consultant – Cardiovascular and Thoracic Surgery

Keywords Paravalvular leak closer (PVL), Mitral regurgitation (MR), Device closure procedure (DCP), Amplatz vascular plug device (AVP II)

Abstract: Paravalvular leak (PVL) is more commonly experienced after surgical valve replacement (mitral/aortic). Post-surgical valve replacement there is a 5-15% chance of developing a paravalvular leak.[1] Moderate to severe paravalvular leak leads to congestive heart failure, haemolytic anaemia or both. Patients may require repeated admission to the hospital for heart failure or blood transfusions/ iron injections. Moderate to severe paravalvular leak needs to be addressed either surgically or by percutaneous device closure procedure (DCP). [2,4]

A 71 gentleman man diabetic, hypertensive, hypothyroidism, ischemic heart disease, chronic atrial fibrillation, anaemic presented with symptoms of heart failure like breathlessness NYHA grade |||, cough, swelling over feet and easy fatiguability- unable to do routine activities. The patient was admitted four times in the past 6 months for heart failure treatment.

The patient had undergone coronary bypass surgery and mitral valve surgery in 2008(Mechanical 27mm st. Jude) - 13 yrs ago. Two coronaries were granted at that time. He showed signs and symptoms of congestive heart failure. On 2 D echo examination showed moderate paravalvular mitral regurgitation(grade2/3) at 11 0 clock position, anteroseptal wall dyskinetic, inferior wall hypokinetic, ejection fraction 40%. The patient was counselled in detail about the need to treat existing paravalvular leaks either surgically or DCP and the risk involved. The general anaesthesia technique was used for PVL DCP. General Anesthesia induction, intubation and extubation have potentially adverse effects on poor heart function(HF) in a raised creatinine renal status. Keeping periprocedural complications in mind, high-risk consent was obtained. General anaesthesia was given, 3D echo preliminary examination done, femoral vein cannulated, the transseptal puncture was done, steerable Aegilis sheath was used to place AVPII device across PVL.thers was no mitral regurgitation post device placement. The patient was extubated on the table. A neurological assessment is done, patient shifted to ICU.

Conclusion: Paravalvular leak (PVL) is more commonly experienced after surgical valve replacement (mitral/aortic). Moderate to severe paravalvular leak needs to be addressed either surgically or by percutaneous device closure procedure. (DCP). AVPII device used to close moderate paravalvular leak at 11 o'clock position of the prosthetic mitral valve. DCP was completed successfully under general anaesthesia. The patient's hemodynamics improved and showed signs of immediate recovery. The patient met his family post-procedure and spoke with them. The patient was discharged home on day 4.

INTRODUCTION:

Paravalvular leak (PVL) is more commonly experienced after surgical valve replacement (mitral/aortic) or transcatheter aortic valve replacement. PVL occurs when there is a permanent defect between the native annulus and prosthetic valve frame. The incidence of PVL after surgical valve replacement ranging from 2-10% in the aortic position, 7-17% in the mitral position. [1,11] The majority of PVL are crescent, oval or roundish-shaped and their track can be parallel, perpendicular or serpiginous. PVL is most commonly observed with mechanical valves followed by bioprosthetic valves. Post-surgical valve replacement there is a 1-5% chance of developing a severe paravalvular leak. The factors that predispose to PVL occurrence are tissue friability, infections, chronic steroids or annular calcification.

Mild paravalvular leak remains asymptomatic. Moderate to severe paravalvular leak leads to congestive heart failure, haemolytic anaemia, or both or infective endocarditis. Patients may require repeated admission to the hospital for heart failure or blood transfusions/ iron injections. Moderate to severe paravalvular leak needs to be addressed either surgically or by percutaneous device closure procedure (DCP). Reoperation is the first-choice procedure when PVL coexists with significant dysfunction or mechanical instability of the prosthetic valve, need for by-pass surgery (CABG), and infectious endocarditis. Surgical PVL closure is a redo surgery hence mortality risk is around 15-20%. After redo surgery for PVL closure, 1 yr, 5 yr, 15 yrs survival was 83%, 62% and 16% respectively. Death was usually due to heart failure and cardiogenic shock. Reoccurrence of PVLwas seen at 1 yr and 5 yrs in 21% and 18%, underwent redo-redo surgery for same. [2,13]

PVL device closure procedure using various devices(Occlutech, amplatz vascular plug family, II, III, IV, Amplatzduct occluder ADO II & III,



Amplatz atrial septal occluder ASO, VSD occluder (AMVSDO) available in the market will have a lesser degree of mortality and morbidity, the risk to life < 5 %.[11,22] The novelty requires thorough pre-procedure evaluation including detailed imaging of anatomy by CT heart and multi planner 3 D reconstruction of TEE images with colour doppler and well-trained team approach in an experienced centre. 4D flow MRI, another novel imaging could be a complementary imaging modality that could assess both structural and hemodynamic aspects of the heart.[3] These procedures are done under GA, fluoroscopic and multi planer 3-D TEE guidance and adequate anticoagulation achieved with intravenous heparin.

Case Report

We present the case of 71 yrs gentleman man, diabetic hypertensive, hypothyroidism, ischemic heart disease, chronic atrial fibrillation, anaemic presented with symptoms of heart failure like breathlessness NYHA grade III, cough, swelling over feet and easy fatiguability- unable to do routine activities. The patient was admitted four times in the past 6 months for heart failure treatment.

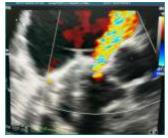
The patient had undergone coronary bypass surgery and mitral valve surgery in 2008 (Mechanical 27mm st. Jude) - 13 yrs ago. Two coronaries were granted at that time.

On examination, the general condition is cachexic, the pulse is law volume, blood pressure is 90/50 mm of hg, jugular venous pressure is raised, pallor, pedal oedema is present. On respiratory system examination basal creps and wheezing, respiratory rate 20-22 per minute. On cardiac examination, a pan-systolic murmur is present in the left sternal border.

Blood investigation as follows, complete blood count- Hemoglobin-9.4mg/dl, WBC count 9890/cumm, platelets-4,84,000, S. creatinine-1.78, K-4.52, prothrombin time-18.90/11.70 control, INR-1.63, S. bilirubin total -1.38 hiigh, direct bilirubin-43 high, LDH-1088 (,230=n)

Chest x-ray examination showed pulmonary congestion and cardiomegaly and the prosthetic mitral valve in situ.

On 2-Echo examination-there is moderate paravalvular mitral regurgitation (grade2/3)proportion of regurgitant area to the circumference of the prosthesis 10-20%, single regurgitant jet located anterolaterally between 10 and 11 o'clock measuring .7X.4 mm, mechanical prosthetic valve leaflets have brisk normal motion. regurgitant volume20 % of actual flow passing through the mitral valve, the inferior septum and inferior wall, is hypokinetic. Anteroseptum and apical anterior wall dyskinetic and scarred. There is severe dilatation of left atrium 87mm, left ventricular ejection fraction 40%, right ventricular ejection fraction-40-45%, PAP 68/28 mm of Hg, no clot, vegetation and pericardial effusion. Right ventricular TAPSE is 9 mm.



Echo shows paravalvular leak at 11 view paravalvular leak at 11 oclock oclock position



Fig No 1:. shows Color Doppler 2 D Fig No 2:shows 3 D Echo Left atrial position

Continuous-wave doppler assessment showed peak and mean gradient across mitral valve 20 mm of hg/5 mm of hg, MVOA:2.80 cm2-3.35cm2, moderate paravalvular regurgitation, regurgitant jet located anterolaterally. There is no intravalvular regurgitation. Left atrum size 87 mm. IVS basal mid and apical dyskinetic.

Left ventricular segmental function diagram (Heger et al method) showed intraventricular septum base apical and mid segments dyskinetic, inferior wall hypokinetic, Antero-apical wall dyskinetic and scarred

Lumber spine showed anterior spondylolisthesis is seen in L4-5, changes in arthropathy seen in the apophyseal joint.

Coronary angiography showed that all grafts functioning well. PAP: 68/28 mm of Hg, left renal artery shows 90% stenosis, left vertebral artery shows 90% stenosis. RA shows a mean pressure of 13 mm of Hg.

The patient was on diuretics, beta-blocker, thyronorm, statins, warfarin (oral anticoagulant) and multiple vitamins. The patient was counselled in detail about the need to treat existing paravalvular leak either surgically or DCP. The risk involved with both procedures were disclosed. Redo surgical closure of PVL had a 15-20% risk to life.DCP being a less invasive alternative had less than 5% risk. After detailed multiple counselling by

team members, the patient gave consent for DCP. The general anaesthesia technique was used for PVLDCP.

General Anesthesia induction, intubation and extubation have potentially adverse effects on poor heart function(HF)and in a raised creatinine renal status. These patients may develop pulmonary oedema, arrhythmias, stroke, vascular complications or cardiac



Preplacement





Fig No 3. shows AVP || device Fig No 4: A)Steerable sheath directed across PVL; B)Device in situ on real time 3 D echocardiography of mitral prosthetic valve (surgeon's veiw)

tamponade during the procedure or any obstruction or interference with existing valve functioning requiring emergency redo cardiac surgery. Post-procedure risks are acute kidney injury, ventilator-dependent respiratory failure. Keeping periprocedural complications in mind Anestgesiologist has to be prepared to deal with them on an emergent basis. Hence experienced Anestgesiologist's expertise should be utilised for the best outcome in these procedures.

High-risk anaesthesia and procedure consent was obtained. The patient was transferred to the cath lab for PVL DCP. Under local anaesthesia, a wide bore 16 G peripheral line is established. Ringer lactate fluid started at 30 ml/hr. Standard monitoring (ECG- 5 lead, pulse oximetry, NIBP, ETCO2) were set up. Invasive arterial and central lines were inserted under local anaesthesia. Noradrenaline 8 mg/50 ml NS and NTG50mg/ 50 ml infusion kept ready. Emergency medicines atropine, adrenaline 1;1000,ephedrine 6 mg/cc , xylocard, avil,hydrocortisone 100 mg kept ready. Single-dose antibiotics were given before the procedure.

General anaesthesia was administered using Midazolam, fentanyl, etomidate and atracurium. 8.0 number endotracheal tube was inserted and maintained with oxygen, air and sevoflurane anaesthesia. Intermittent muscle relaxant blouses were given. After induction 3-D trans oesophageal Echo probe was inserted for detailed guidance throughout the procedure. Intravenous vascular dose heparin 1 mg/kg givento achieve ACT>=250 sec.

PROCEDURE:

The right femoral vein was cannulated with 6.5 F sheath, TEE guided septal puncture at mid fossa was done.8.5F Agilis sheath was advanced into the left atrium. The defect was crossed with Terumo wire. The MP catheter was advanced into the left ventricle. the course of the catheter was confirmed on TEE. Hemodynamic assessment is done. Amplatz stiff wire was placed in the left ventricle and a 10x7mm AVP II device was placed across the defect through steerable sheath under real-time 3D echocardiographic assessment (surgeon` view). TEE showed no mitral regurgitation at the site of the defect. Pre-procedure PAP was 30/17(23) mm of Hg. Post procedure PAP was 23/17(20) mm of Hg. Left atrial pressure and v wave were reduced. [14] The sheath was removed. The femoral venous puncture was compressed.

The patient was extubated on the table. The patient was oriented, breathing spontaneously and responding to verbal commands. oxygen levels were maintained.

A neurological assessment is done. The patient was shifted to ICU for observation for a day. The patient was discharged to home on day 4.10 days follow up, the patient was completely relieved of breathlessness.

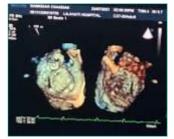


Fig No 5 Multiplaner 3 D echo showing amplatz vascular plug II in situ with free movements of mechanical prosthetic mitral leaflets (open and closed leaflets)

Fig No 6: fluroscopic image above arrow shows amplatz vascular plug II device in anterolateral position and below arrow shows prosthetic mitral valvering.

The paravalvular leak is common after surgical valve replacement or transcatheter aortic valve

DISCUSSION:

replacement. Mild paravalvular leak remains asymptomatic. Moderate to severe PVL manifest as heart failure- NYHA class III-IV breathlessness and hemolytic anaemia or both or infective endocarditis. Hence It warrants correction either surgically - redo surgery or by percutaneous DCP. Surgical correction of PVLis associated with 15-20% mortality risk. Various studies of percutaneous DCP states the risk of mortality up to 5%.DCP is associated with significantly lower mortality and morbidity compared to surgical PVL closer.

Multiple modalities have been used to diagnose PVL accurately. The primary investigation, 2-D Echocardiography and doppler assessment will offer valuable information related to hemodynamic consequences of PVL, such as valve function, heart chamber size and function, and pulmonary artery pressure. However, grading and quantification of PVL using conventional echocardiographic parameters, such as vena contract, or the effective regurgitant orifice area and regurgitant volume measured by the proximal iso velocity surface area method, is a considerable challenge because PVL is usually observed as eccentric regurgitation. The novelty requires thorough pre-procedure evaluation including detailed imaging of anatomy by CT heart and multi planner 3 D reconstruction of TEE images with colour doppler and well-trained team approach in an experienced centre. 4D flow MRI, another novel imaging could be a complementary imaging modality that could assess both structural and hemodynamic aspects of the heart. [20]

Various devices available in markets have been used to occlude leaks. Occlutech PLD is one such device specifically designed for PVL closure. Most commonly used are Amplatz vascular plug family –II, III, IV devices. [11,22] There are many case reports of using Amplatz muscular VSD occluder (AMVSDO) or amplatz patent ductus arteriosus occluder device for PVL closure. [19]

In our case, PVL was a single, oval in shape, situated anterolaterally at 11 O clock position. The transseptal approach was selected. The defect was 7×4 mm. Amplatz vascular plug device II of the diameter of $10 \text{mm} \times 7 \text{mm}$ was chosen. Through the femoral vein, the septum was a puncture in the mid fossa with the needle. Amplatz wire was crossed across PVL and placed in LV. Device amplatz vascular plug II was



mounted on a Steerable sheath and was placed across PVL. The device was released into the defect. Post device placement there was no MR. Hemodynamics improved. Mechanical prosthetic valve leaflets were functioning normally.

In the case of mitral PVL, antegrade transseptal or retrograde transapical approach can be used. Defect location is in the lateral wall, transseptal approach is used. Defect on the posteromedial or posterolateral side adjacent to interatrial septum, the transapical approach is used. [9,18] In the transapical approach epicardial ultrasonographically guided left minithoracotomy incision is taken. Two U mattress sutures were placed in the anterolateral portion of the heart, close to the second diagonal coronary artery. Left ventricular access was achieved, through defect wire was placed in LA and devices are delivered. Peratrial approach has also been used. The probe-assisted delivery system is shorter, simpler, and more cost-effective than that of the other approaches. Aiming at safe obliteration of leak without compromising the function of the prosthetic valve leaflets. Different techniques were used including single device placement, simultaneous (double wire) deployment, sequential (anchor wire) deployment, and sequential deployment using an arteriovenous rail. In the patient with PVL in aortic as well as mitral position, both leaks were closed with devices in two different settings. These devices may impinge on valve disk and interfere or obstruct normal valve functioning or may slip and embolism requiring emergency redo surgery. Other complications associated with DCP are vascular injury requiring blood transfusions, hemothorax, acute kidney injury(AKI), stroke or transient ischemic attack(TIA), cardiac tamponade or VDRF(ventilator-dependent respiratory failure.

The ideal PVL closure device should meet the following criteria:(a) should fit into 'irregular' defects, (b) easy deliverability, (c) be repositionable and retrievable, (d) avoid interference with prosthetic valve leaflets, (e) should close defect completely (f) have a low risk of embolisation or dislodgement and (g) should be non-thrombogenic [19,20]

CONCLUSIONS:

The percutaneous device closure procedure for the mitral paravalvular leak (PVL) is effective in improving symptoms and reducing the need for mitral valve reoperation. [11,18] In patients undergoing percutaneous mitral PVL closure, successful reduction of the PVL to mild or less is associated with significant improvement in short- and mid-term survival. [13,18] Longer time from mitral valve replacement surgery to PVL closure, severe pulmonary hypertension, and the presence of multiple leaks predict inadequate PVL reduction with percutaneous techniques. [4]

REFERENCES:

- Dr Grzegorz Smolka, FESC Mr Wojciech Wojakowski, et al. Paravalvular leak important complication after implantation of the prosthetic valve, An article from the E-Journal
 of the ESC Council for Cardiology Practice, Vol. 9, No 8 08 Nov 2010
- Sameh M Said 1, Hartzell V Schaff 1, Kevin L Greason et al ;Reoperation for mitral paravalvular leak: a single-centre experience with 200 patients. Interact Cardiovascular Thorac Surg 2017 Nov 1;25(5):806-812.doi:10.1093/icvts/ivx222
- Jah Yeon Choi 1, Young Joo Suh 2, Jiwon Seo et al; Structural and Functional Characteristics of Mitral Paravalvular Leakage Identified by Multimodal Imaging and Their Implication on Clinical Presentation; J Clin Med 2021 Jan 10; 10(2):222. DOI: 10.3390/jcm10020222.PMID: 33435160,PMCID: PMC7826927
- 4. Alkhouli, M.; Zack, C.J.; Sarraf, M.; Eleid, M.F.; Cabalka, A.K.; Reeder, G.S.; Hagler, D.J.; Maalouf, J.F.; Nkomo, V.T.; Rihal, C.S. Successful Percutaneous Mitral Paravalvular Leak Closure Is Associated With Improved Midterm Survival. Circ. Cardiovasc. Interv. 2017, 10. [CrossRef]
- 5. Garcia, E.; Arzamendi, D.; Jimenez-Quevedo, P.; Sarnago, F.; Marti, G.; Sanchez-Recalde, A.; Lasa-Larraya, G.; Sancho, M.; Iniguez, A.; Goicolea, J.; et al. Outcomes and predictors of success and complications for paravalvular leak closure: An analysis of the Spanish real-world paravalvular Leaks closure (HOLE) registry. EuroIntervention 2017, 12, 1962–1968. [CrossRef] [PubMed]
- 6. Giblett, J.P.; Rana, B.S.; Shapiro, L.M.; Calvert, P.A. Percutaneous management of paravalvular leaks. Nat. Rev. Cardiol. 2019, 16, 275–285. [CrossRef] [PubMed]
- 7. Sorajja, P.; Cabalka, A.K.; Hagler, D.J.; Rihal, C.S. Long-term follow-up of percutaneous repair of paravalvular prosthetic regurgitation. J. Am. Coll. Cardiol. 2011, 58, 2218–2224. [CrossRef] [PubMed]
- 8. Hein, R.; Wunderlich, N.; Robertson, G.; Wilson, N.; Sievert, H. Catheter closure of paravalvular leak. EuroIntervention 2006, 2.
- Vinod H. Thourani, MD, Colleen M. Smith, BA, Robert A. Guyton, MD, Peter Block, MD, David Liff, MD, Patrick Willis, MD, Stamatios Lerakis, MD, Chesnal D. Arepalli, MD, Sharon Howell, RDCS, Bryon J. Boulton, MD, James Stewart, MD, and Vasilis Babaliaros, MD, Repair of Prosthetic Mitral Valve Paravalvular Leak Using an Off Pump Transapical Approach. Ann Thorac Surg; 2012;94:275–78.
- S. Johnson, T. Ho; Multiple paravalvular leak closures. 34TH EACTA ANNUAL CONGRESS ABSTRACTS / Journal of Cardiothoracic and Vascular Anesthesia 33 (2019) S105-S139, S-113.
- 11. Li Hongxin, MD, Guo Wenbin, MD, Hai-Zhou Zhang, MD, Fei Liang, MD, Gui-Dao Yuan, MD, Zeeshan Farhaj, MBBS, and Jun Zhang, MD: Peratrial Device Closure of Different Locations of Mitral Paravalvular Leaks Ann Thorac Surg; 2018;105:1710–6.
- 12. Patrick A. Calvert, PhD David B. Northridge, MB Iqbal S. Malik, PhD Leonard Shapiro, MD; Percutaneous Device Closure of Paravalvular Leak Combined Experience From the United Kingdom and Ireland Circulation. 2016;134:934–944. DOI: 10.1161/CIRCULATIONAHA.116.022684
- James W. Lloyd MD, Charanjit S. Rihal MD, MBA, Guy S. Reeder MD, Rick A. Nishimura MD, Acute invasive hemodynamic effects of percutaneous mitral paravalvular leak closure; Cardiac catheterisation and intervention, Issue 5, volume 90,851-858.
- 14. V.K. Trehan, an M.S.K. Subhendu,b, V. Chaturvedi,b B.N. Pandit,c and M. Goyalc et al; Paravalvular leak closure with two large-size devices. Indian Heart J. 2014 Jan-Feb; 66(1): 91–94. Published online 2013 Dec 26. DOI: 10.1016/j.ihj.2013.12.033 PMCID: PMC3946467, PMID: 24581103.
- 15. M. Boochi Babu, 1 D. Rajasekhar, 1 V. Vanajakshamma, 1 A. Chandra, 2 M. Hanumantha Rao, 3 M.L. Sreenivasa Kumar 1 et al; Percutaneous transcatheter closure of a mitral paravalvular leak via anterograde approach without arteriovenous loop in a patient with double valve replacement. J Clin Sci Res 2013;2:174-8.
- 16. Ankit Garg, Sushil Azad, Sitaraman Radhakrishnan: Percutaneous paravalvular leak closure with their outcomes: A single-centre experience. Annals of Cardiac Anaesthesia | Volume 24 | Issue 3 | July-September 2021
- 17. Raghuram Palaparti,1 Kothandam Sivakumar,2 Uma Maheswar KL,2 Sreeja Pavithran2: Transcatheter Device Closure of Paravalvular Leaks: A Single-Center Experience. Journal of the American college of cardiology, VOL. 74, NO. 13, SUPPLB, 2019
- Oreoluwa Oladira, Gabriel Areoye, Adeolu O. Oladunjoy et al; Closure of a Prosthetic Mitral Valve Paravalvular Leak Using a Ventricular Septal Defect (VSD) Amplatzer Occluder Device. Am J Case Rep, 2021; 22: e928003 DOI: 10.12659/AJCR.928003.
- K. Kalogeras, K. Ntalekou, K. Angeli et al., Transcatheter closure of paravalvular leak: Multicenter experience and follow-up, Hellenic Journal of Cardiology, https://doi.org/10.1016/j.hjc.2021.02.002
- 20. I.Cruz-Gonzalez 1 J. C. Rama-Merchan 1, 2 J. Rodríguez-Collado 1 J. Martín-Moreiras 1 A. Diego-Nieto 1 M. Barreiro-Pérez 1 P. L Sánchez: Transcatheter closure of paravalvular leaks: state of the art: Neth Heart J (2017) 25:116–124. DOI 10.1007/s12471-016-0918-3.
- 21. Ignacio Cruz-Gonzalez,* Juan Carlos Rama-Merchan, Antonio Arribas-Jimenez, Javier Rodriguez-Collado, Javier Martin-Moreiras, Manuel Cascon-Bueno, and Candido Martin Luengo:Paravalvular Leak Closure With the Amplatzer Vascular Plug III Device: Immediate and Short-term Results; Rev Esp Cardiol. 2014;67(8):608–614.
- 22. Sameer Gafoor, Jennifer Franke, Stefan Bertog, Simon Lam, Laura Vaskelyte, Ilona Hofmann, Horst Sievert and Predrag Matic: A Quick Guide to Paravalvular Leak Closure, review article, Interventional Cardiology Review, 2015;10(2):112–7

CASE REPORT II: ANAESTHESIOLOGY

Anaesthetic challenges in management of a Small Bowel Neuroendocrine Tumour: A case report

Dr. Samidha Waradkar Thakur, DA, DNB Anaesthesiology, PGDMLS, Consultant Anaesthesiology Dr. Prakash Padamukhe, Primary DNB Anaesthesiology

Abstract

Small bowel neuroendocrine tumours (NET) are rare secreting tumours of the enterochromaffin or Kulchitsky cells. (1.2) Hormone release can be caused by stress, hypoxemia, hypothermia, hypo or hypertension, pain, induction of anaesthesia, tumour manipulation and pharmacological agents. (1)

This case report discusses the various perioperative anaesthetic challenges and considerations during peri-operative management of NET.

Introduction

Carcinoid tumours were first described in 1888 by Lubarsch. (3) Hormones such as serotonin, histamine, bradykinin, prostaglandins, and chromogranin-A secreted into the systemic circulation can lead to a carcinoid syndrome. (1) It's characterized by symptoms such as flushing, diarrhoea, abdominal pain, bronchospasm, cardiovascular changes. (1)

Case report

53 yrs old hypertensive female presented with Malena, loss of appetite, headache, light headedness. She was diagnosed to have small bowel NET and posted for exploratory laparotomy.

On preoperative evaluation, patient was vitally stable. All her routine investigations, ECG, 2D Echo, Chest X-ray were found to be normal.

PET CT showed low grade metabolically active concentric mural thickening in the ileum with a well-defined mesenteric nodular lesion suggestive of neuroendocrine etiology. (fig. 1.)

Gallium-68 DOTATOC scan showed evidence of high-grade somatostatin receptor expression in the same areas.

Adrenaline values at 187 pg/ml and of Noradrenaline 1137.6 pg/ml were elevated. Chromogranin A levels were also high.

Cardiologist fitness was confirmed. Patient was started on T. MetXL 12.5 OD, T. Labetalol 100mg BD.

On the day of surgery, patient was premedicated with T Pan D 40mg, IV Avil 2cc, IV Hydrocortisone 100mg.

Intraoperatively, after connecting all routine ASA monitoring, an epidural catheter was inserted at T11-12 level under local Anaesthesia (LA). Patient was sedated using IV Midazolam 1mg, IV Fentanyl 100mcg, IV Ondensetron 8mg and IV Dexmedetomidine 1mcg/ml titrated infusion was initiated. Right radial artery was secured under LA. Patient was induced using IV Propofol 50mg, IV Cisatracurium 50mg (no histamine releasing drugs) and airway was secured without any sequelae. Right IJV was then cannulated using 7Fr triple lumen catheter for CVP monitoring and administration of cardiac supports.

Monitoring of ECG, SpO2, Invasive BP, EtCO2, Temperature, CVP, Urine output, ABG, MAC, Paw was done continuously. Maintenance of Anaesthesia was done by Air:O2=50:50, Sevoflurane 1-2%, Boluses of Cisatracurium, epidural infusion of 0.25% Bupivacaine Fig. 1. PETCT image showing enhancement due to NET @3-5ml/hr, IV Dexmedetomidine infusion. BP was maintained at

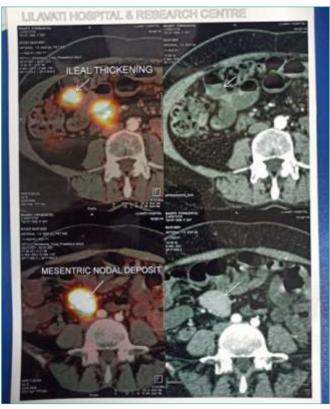








Fig. 2. Multipara monitor on Anaesthesia workstation showing intraoperative monitoring.

Fig. 3. Intraoperative picture showing invasive lines, inline fluid warmer and infusions.

MAP of 60-70 mmHg using Phenylephrine boluses of 20-40mcg.

The surgeon was quick to identify the diseased segment and the blood supply to the NET was sealed off, thus preventing any major adverse events

Total intraoperative duration of surgery and Anaesthesia was 6.5 hours. Barring few episodes of hypotension which responded to fluid and phenylephrine boluses, the surgery was largely uneventful. At the end, patient was reversed and extubated smoothly and epidural infusion was started for postoperative analgesia. Patient was shifted to ICU for further management.

Discussion

NET of small intestine are the most common causes of the carcinoid syndrome. (4) Under electron microscopy, they typically contain numerous membrane-bound neurosecretory granules composed of hormones and biogenic amines. (2)

Serotonin is synthesized by hydroxylation and decarboxylation of tryptophan. Serotonin can produce both vasoconstriction and vasodilatation, thus resulting in hyper or hypotension. $^{(3)}$

Carcinoid heart disease occurs in more than half of the patients with the carcinoid syndrome. (4) For anaesthetic purposes, patients with carcinoid tumours should be regarded as suffering from a multi-system disease. (2)

Morphine and atracurium have most potential for unwelcome histamine release and could be avoided. Suxamethonium has been implicated in the release of peptides from the liver as a consequence of depolarization-induced fasciculations. (2) Elevated levels of serotonin can lead to hyperglycemia. So blood glucose level should be monitored and controlled. (3)

Norepinephrine has been shown to activate kallikrein in the tumour and can even lead to the synthesis and release of bradykinin resulting paradoxically in further vasodilatation and worsening hypotension. (2) Administration of small doses of phenylephrine has been found helpful.

Vasoactive hormone release intra-operatively is best treated with intravenous boluses of 20–50 µg of octreotide, titrated to haemodynamic response. Vasopressin is an alternative vasoconstrictor. (2)

Conclusion

Carcinoid syndrome is a challenge for the Anaesthesiologist due to its unpredictable clinical manifestations. Good communication between an Anaesthesiologist, surgeon and endocrinologist is necessary for good outcome.

REFERENCES:

- 1. Intraoperative carcinoid syndrome during small-bowel neuroendocrine tumour surgery. Myrtille Fouche et al, Endocrine Connections (2018) 7, 1245-1250
- 2. Carcinoid: the disease and its implications for anaesthesia Bruce Powell, MRCP FRCA et al, Continuing Education in Anaesthesia Critical Care & Pain, Volume 11, Issue 1, February 2011, Pages 9–13
- 3. Carcinoid tumours: Challenges and considerations during anesthetic management. Sukhminder Jit Singh Bajwa Year: 2015 | Volume: 42 | Issue: 3 | Page: 132-137
- 4. ENETS Consensus Guidelines for the Standards of Care in Neuroendocrine Tumors: Pre- and Perioperative Therapy in Patients with Neuroendocrine Tumors. Gregory Kaltsas et al, Neuroendocrinology. 2017 Sep; 105(3): 245–254

CASE REPORT III: ANAESTHESIOLOGY

Total Lung Lavage using THRIVE for pre-oxygenation and oxygen insufflation during intubation.

Dr. Vaibhavi Baxi, DA, FCPS, DNB, Consultant – Anaesthesiology **Dr. Prahlad Prabhudesai,** MD, DNB, FCCP, Consultant – Chest Medicine **Dr Abha Mahashur,** MBBS, DNB (Respiratory Diseases),TB IDCCM, Consultant – Chest Medicine

Abstract:

Pulmonary alveolar proteinosis (PAP) is a rare disease characterized by deposition of lipoproteinaceous material in the alveoli secondary to abnormal processing of surfactant by macrophages. Total-lung lavage (TLL); a gold standard management for this disease entails washing out the proteinaceous material from the alveoli and re-establish effective oxygenation and ventilation. Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE), utilizes apnoeic oxygenation to extend the apnoeic window and delay rapid de-saturation common in these cases; thus allowing for safer intubation. We would like to share our experience of two cases of TLL for PAP in which we used THRIVE for pre-oxygenation and apnoeic oxygenation during intubation. Both patients were young with low baseline oxygen saturations (SpO2) and partial pressure of oxygen in blood (pO2) on air. Hence the decision was taken to pre-oxygenate them using THRIVE and to also use THRIVE during intubation to improve their tolerance to apnoea during intubation. When used for pre-oxygenation and oxygen insufflation during intubation THRIVE prolongs the tolerable apnoea time and delays de-saturation during intubation.

To our knowledge this is the first case report demonstrating the use of THRIVE in a case of TLL in literature.

Keywords:

Pulmonary alveolar proteinosis (PAP), Total Lung Lavage (TLL), Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE).

Introduction:

Total lung lavage (TLL) is a therapeutic procedure performed for treatment of Pulmonary alveolar proteinosis (PAP), a rare disease with prevalence of approximately 1-2 per million1. In PAP, there is accumulation of phospholipo-proteinaceous material in the alveoli causing pulmonary gas exchange impairment. [1] A thorough TLL results in significant improvement of symptoms and radiographic appearance of lungs[1]. Lung isolation is essential for TLL and oxygenation with isolation in diseased lung is often a challenge.

Defective clearance and accumulation of surfactant in alveoli in PAP results in poor gas exchange and reduced oxygen reserve; thus significantly reducing the tolerable apnoea time in patients. We used the Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE) technique of delivering up to 30 L/min of heated and humidified air-oxygen mixture up to 100% Fraction inspired Oxygen(FiO2) for pre-oxygenation and 70 L/min of same for apnoeic oxygenation during intubation. This helped in maintaining oxygen saturation and gave extended apnoea duration for intubation.

In this case report we review the safety and efficacy of THRIVE for two patients undergoing TLL.

Case 1

A 35-yrs old female with body mass index (BMI) 21.9kg/m2 diagnosed with PAP had an oxygen saturation (SpO2) of 82% on room air. Serial Lung Lavage of both lungs five days apart, (left lung (with more severe disease) followed by right lung); was planned under general anaesthesia. A radial arterial line was placed prior to intubation for hemodynamic monitoring and for serial arterial blood gases (ABG). Preinduction ABG on air demonstrated a pH7.47, pCO2- 30.9, pO2-46 and bicarb24.1 m mol/l.

Pre-oxygenation was carried out by THRIVE unit. Oxygen was administered prior to induction at 30L/min with an FiO2 of 100% till Saturation improved from 80% at room air to 97%. Anaesthesia induction utilized fentanyl and propofol and cis- atracurium. After mask ventilation for three minutes with 1% sevoflurane on 100% oxygen, THRIVE nasal cannula was reapplied. The flow rate was increased to 70 L/min with an of FiO2 100% during the period of intubation. Intubation was successful in the second attempt with a 35Fr. left double lumen endotracheal tube (DLT). There was no fall in oxygen saturation during intubation. Position of DLT with adequate isolation of ventilation was confirmed with fibreoptic bronchoscope (FOB).

The right lung was ventilated with clamp on the ventilating port of left side of the DLT. Through a Y connector in the left port of DLT, inlet and outlet tubings were connected for the lavage fluid. (Fig. no. 1) The left lung was lavaged with a total of 6.9 L of warm (370 C) sterile warm



normal saline in 500 ml increments every 2min 30 seconds with a return of 7.4 L. Each 1 litre of normal saline contained 500 IU of Heparin and 25 ml of Sodium Bicarbonate. After each 500 ml aliquot, the physiotherapist gave manual and chest physiotherapy in different positions of the patient to aid in dislodgement of secretions. Following which the fluid would be drained out through the outflow tube in glass bottles and be measured. Initial lavage fluid was opalascent and milky white. The final bottle of lavage fluid collected was thin in consistency and clear.

Anaesthesia was maintained using sevoflurane, fentanyl and cis- atracurium. We encountered high airway pressure upto 50 mm of Hg and fall in SpO2 to 70% intermittently during the procedure, which was managed by 100% FiO2 and suction of the ventilated lung. EtCO2 went up to 48 mm Hg during the procedure. At the end of the procedure which lasted about 2.5 hrs, the DLT was exchanged to a single lumen ET. Patient was then extubated once all haemodynamic parameters and gases were within normal limits. After extubation patient was put on THRIVE again with 30 Lmin-1 with SpO2 95%.

Postoperatively patient was shifted to ICU and her ventilation was assisted with bi-level positive airway pressure (BIPAP) overnight. With FiO2 0.45 the ABG showed a pO2 of 140 and pCO2 of 43 mmol/l. Remarkable radiological clearance of left lung opacities along with corresponding improvement in patient's breathing pattern was seen next day. Right lung lavage was done uneventfully after a gap of five days in a similar pattern.

Case 2

A 40-yrs female BMI 28.14kg/m2; diagnosed with PAP had room air SpO2 89%. Similar to previous case, serial lung lavage of both lungs five days apart (right lung followed by left) was planned under general anaesthesia. Pre-induction ABG with 2 L/min of O2 via nasal prongs revealed a pH of 7.395, pCO2 of 40.5 mm Hg, pO2 of 96mm Hg with bicarbonates(std) of 24mmol/l.

Pre-oxygenation was again conducted via THRIVE at 30L/min with FiO2 100% until SpO2 improved from 90% to 99%. After induction and mask ventilation for three minutes, the THRIVE nasal cannula was re-applied. The flow rate was increased to 70 L/min with FiO2 of 100% during the period of intubation of trachea with a 35Fr. DLT. Total duration needed was 5 minutes due to small laryngeal inlet and short length of trachea. Position of DLT with adequate isolation was confirmed clinically and with fibreoptic bronchoscope. Lung lavage of right lung was done with seven litres of prewarmed saline in 500ml aliquots in a similar way as the previous patient using a Y connector in right channel of DLT. We encountered airway pressures of upto 36 mm of Hg and fall in SpO2 up to 95% intermittently, which was managed by 100% FiO2 and

intermittent suction of ventilated lungs. At the end of 2 hour procedure patient was extubated and put on THRIVE with 30 L/min with SpO2 100%.

Postoperatively patient was shifted to ICU and her ventilation was assisted with BIPAP overnight. Significant improvement in ventilatory exchange was seen on post operative day one. For FiO2 of 40%, now PO2 was 143mm Hg with significant clinical improvement in ease of breathing. The left lung was lavaged uneventfully in a similar way after a gap of five days.





Discussion:

PAP is characterized by the accumulation of phospholipoproteinoceous material in the alveoli that stains positive on PAS stain and its accumulation leads to compromised gas exchange.^[2,3]. In HRCT of the chest, reticulations superimposed with ground glass opacities form a crazy paving pattern which is characteristic of PAP.^[3,4] The most effective palliative therapy for PAP is the mechanical removal of the proteinaceous material via TLL^[1,5,8].

THRIVE is the delivery of trans-nasal humidified oxygen by high-flow nasal prongs. (Fig. no.2) The circuit consists of an air-oxygen blender attached to a flow meter, which controls the concentration and flow of oxygen delivered. It can generate up to 100% FiO2 at 70 L/min, with a positive end-expiratory pressure of 7.4 cm H2O. Traditionally, pre-oxygenation has been performed by mask ventilation with 100% oxygen with aim to displace nitrogen in the lungs and create an oxygen reservoir. As long as a patent airway exists, the difference in rate of alveolar oxygen removal and carbon dioxide excretion generates a negative pressure gradient that pulls oxygen into the lungs. This phenomenon is known as aventilatory mass flow [9,10]. The concept of THRIVE revolves around extension of the apnoeic window—the small time frame following anaesthetic induction for securing a definitive airway^[10]. The application of a high flow nasal delivery system provides large volumes of oxygen to the lungs and creates an oxygen reservoir, prevents entrapment of room air to ensure continual delivery of a high concentration of oxygen and reduces the rate of build-up of carbon dioxide.

Patel and Nouraei used THRIVE for pre-oxygenation in twenty five patients with known difficult airways, and found a mean apnoea time of 17 min with no desaturations below 90%. Furthermore, two patients received THRIVE for the duration of their procedures, with apnoea times of 32 and 65 min [10]. Mir et al studied THRIVE in context with rapid sequence induction in emergency surgery – THRIVE preoxygenation vs face mask preoxygenation. They concluded that THRIVE prolongs the apnoea time significantly and the blood gas profiles were similar in both groups [11]. Lodenius et al in their study of comparison of traditional face mask with THRIVE in emergency rapid sequence induction also concluded that THRIVE could have superior benefits during oxygenation as compared to a face mask [12]. Ansari et al in their study found that post operative use of high flow nasal oxygen contributed to reduced length of hospital stay in elective lung resection surgery. [13]

PAP patients are prone to rapid de-saturation and de-nitrogenation alone is insufficient to counter this. THRIVE provides humidified oxygen with PEEP which prevents air entrapment and alveolar collapse.

Preoxygenation with THRIVE significantly improved the baseline SpO2 and provided us with extended apnoea time for intubation. We also utilized the THRIVE post extubation to help maintain the oxygen saturation. Patients were comfortable with the high flow of nasal oxygen. Postoperatively patients were shifted to intensive care where BIPAP was applied overnight with 40% FiO2 as requested by the pulmonolgist as per their protocol. However there is a potential to utilise THRIVE postoperatively in intensive care as well.

Conclusion:

Our study highlights the benefits of THRIVE in PAP patients by extending the apnoeic window by improving the baseline SpO2 and delaying de-saturation during intubation by continuing oxygen insufflation during the period of intubation.

Clinical message:

THRIVE is a relatively newer modality of oxygen supplementation. TLL though not a common surgery; is very demanding and needs immense planning, counseling and involvement of a big team of pulmonologist, anaesthetist, physiotherapist and intensivist. Patients needing TLL often have bilateral lung disease with poor lung reserve. Lung isolation is essential in such patients and placement and confirmation of the double lumen tube is often time consuming. Use of THRIVE for preoxygenation and during intubation helps in maintaining the oxygen saturation during intubation and extends the tolerable apnoeic duration. Our experience highlights the benefits of THRIVE in PAP patients by extending the apnoeic duration by improving the baseline SpO2 by preoxygenation and delaying de-saturation during intubation by continuing oxygen insufflation during the period of intubation.

We have no conflict of interests.

REFERENCES:

- 1. Hadda V, Tiwari P, Madan K, Mohan K, Gupta N, Bharati SJ et al. Pulmonary alveolar proteinosis: Experience from a tertiary care center and systematic review of Indian literature. Lung India. 2016;33(6):626–634.
- 2. Awab A, Khan MS, Youness HA. Whole lung lavage technical challenges and management of complications. J Thorac Dis. 2017;9(6):1697-1706.
- 3. Borie R, Danel C, Debray MP, Taille C, Dombret MC, Aubier M, Epaud R, Crestani B. Pulmonary alveolar proteinosis. European Respiratory Review. 2011 Jun 1;20(120):98-107.
- 4. Holbert JM, Costello P, Li W, Hoffman RM, Rogers RM. CT features of pulmonary alveolar proteinosis. AJR Am J Roentgenmaol. 2001;176(5):1287–94.
- 5. Shah PL, Hansell D, Lawson PR, Reid KB, Morgan C. Pulmonary alveolar proteinosis: Clinical aspects and current concepts on pathogenesis. Thorax. 2000;55:67–77.
- Rogers RM, Levin DC, Gray BA, Moseley LW., Jr Physiologic effects of bronchopulmonary lavage in alveolar proteinosis. Am Rev Respir Dis. 1978;118(2):255–64.
- 7. Du Bois RM, McAllister WA, Branthwaite MA. Alveolar proteinosis: diagnosis and treatment over a 10-year period. Thorax. 1983;38:360–3.
- Kariman K, Kylstra JA, Spock A. Pulmonary alveolar proteinosis: prospective clinical experience in 23 patients for 15 years. Lung. 1984;162(4):223–31.
- 9. Bartlett RG Jr, Brubach HF, Specht H. Demonstration of aventilatory mass flow during ventilation and apnea in man. Journal of Applied Physiolology 1959; 14: 97–101.
- 10. Patel A, Nouraei SA. Transnasal Humidified Rapid-Insufflation Ventilatory Exchange (THRIVE): a physiological method of increasing apnoea time in patients with difficult airways. Anaesthesia. 2015;70(3):323-329.
- 11. Mir F, Patel A, Iqbal R, Cecconi M, Nouraei SA. A randomised controlled trial comparing THRIVE preoxygenation with facemask preoxygenation in patients undergoing rapid sequence induction of anaesthesia. Anaesthesia. 2017;72(4):439-443.
- 12. Lodenius A, Piehl J, Ostlund A, Ullman J, Jonsson Fagerlund M. THRIVE vs facemask breathing preoxygenation for rapid sequence induction in adults: a prospective randomised non blinded clinical trial. Anaesthesia. 2018;73(5):564-571.
- 13. Ansari BM, Hogan MP, Collier TJ, et al. A randomized controlled trial of high flow nasal oxygen (optiflow)as part of an Enhanced recovery after surgery (ERAS) after lung resection surgery. Ann Thorac surg. 2016;101(2):459-464.

ABBREVIATIONS:

PAP: Protein alveolar proteinosis; TLL: Total lung lavage; THRIVE: Transnasal Humidified Rapid-Insufflation Ventilatory Exchange; FiO2: Fraction of inspired oxygen; BMI: Body mass index; SpO2: Saturation of oxygen; ABG: Arterial blood gas; DLT: Double lumen tracheal tube; FOB: Fibre-optic Bronchoscope; BIPAP: Bi-level positive airway pressure



CASE REPORT IV: CHEST MEDICINE

Bilateral Whole Lung Lavage in Hereditary Pulmonary Alveolar Proteinosis using Extracorporeal Membrane Oxygenation- A case series

Dr. Indu Khosla, MD, DCH, RCPCH Fellow in Ped. Pulmonology, Consultant – Pediatric Chest Medicine

Dr. Prahlad Prabhudesai, MD, DNB, FCCP, Consultant - Chest Medicine

Dr. Satish Kulkarni, DA, MD, (Mumbai), DA (UK), FRCA, Consultant – Anaesthesiology

Dr. Manish Kumar Arya, MD – Pediatrics, Consultant – Pediatrics

Pulmonary alveolar proteinosis (PAP) is a disorder characterized by accumulation of surfactant within alveoli due to either altered production or removal or both leading to increasing respiratory distress and hypoxemic respiratory failure in severe cases. 1 The hereditary form of pulmonary alveolar proteinosis (PAP) due to Colony stimulating factor 2 receptor alpha (CSF2RA) gene mutation is a rare disease with only a few cases reported worldwide.2 We report a case series of two cases of hereditary PAP with a remarkable response to whole lung lavage using extracorporeal membrane oxygenation (ECMO).

Case 1:

4 yrs 8 months old girl born of 3rd degree consanguinity presented with c/o cough, and progressive breathlessness since 3 months. She was hypoxic in the last 2 weeks requiring oxygen. She was admitted twice in the past I year for cough and breathlessness wherein a diagnosis of viral respiratory tract infection was made. Her birth, immunization, developmental and family history were unremarkable. Basic hematology work up done during her previous admission was normal.

Clinical examination on presentation to our hospital revealed a child with failure to thrive, with a weight of 12.1 kgs (<1st centile) and a height of 97 cm (< 3rd centile). She was afebrile, with a pulse of 120/minute and respiratory rate of 46/minute. She had marked subcostal and intercostal retraction and a SpO2 of 93% on 10 litres of oxygen on non rebreathing mask. On systemic examination child had pectus excavatum with bilateral coarse crackles on auscultation, with all systems being normal.

Arterial Blood Gas (ABG) on O2 of 10 Litres/min showed PaO2-104; PaCO2-45.8; pH-7.31; HCO3-22.4 Her thyroid function tests, ECG and 2D echo were within normal limits. HIV was negative. Serum Lactate dehydrogenase (LDH) was raised-694 U/L (140-280 U/L). X-Ray chest showed bilateral reticular infiltrates and CT chest showed diffuse intralobular and interstitial thickening with ground glass opacities (crazy paving pattern) suggestive of Interstitial Lung Disease. Clinical and radiological features were suggestive of Pulmonary Alveolar Proteinosis and further investigations were done to confirm the diagnosis

Granulocyte-Macrophage Colony Stimulating Factor (GM-CSF) auto-antibody test was normal and Serum GM-CSF concentration was raised ruling out autoimmune aetiology. Clinical exome sequencing was positive for CSF2RA gene mutation. Next Generation Sequencing (NGS) showed (Exon 1-5, c deletion, exonic deletion), homozygous, X-linked, suggestive of pulmonary surfactant metabolism dysfunction 4.

With a confirmed diagnosis of Congenital PAP due to CSF2RA gene mutation a therapeutic Whole Lung Lavage (WLL) was planned. In view of the severe respiratory insufficiency and non-availability of appropriate size of double lumen Endotracheal tubes and bronchial blockers for a child of weight 12 kg it was decided to put the child on ECMO and mechanical ventilation to ensure adequacy of oxygenation during the whole lung lavage.



Fig. 1. Chest X-ray showed widespread bilateral reticular shadowing air space disease (Preprocedure)

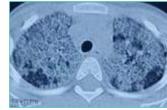


Fig. 2. Computed tomographic scan of the chest showed ground glass opacification and interlobular septal thickening (crazy paying pattern).

PROCEDURE

Patient was intubated with 3.5 mm uncuffed endotracheal (ET) tube and mechanically ventilated. The right Internal jugular vein was cannulated with 16 F double lumen ECMO cannula. Once patient was stabilised on ECMO, lung lavage was started with prewarmed (37*C)0.9% Normal saline in aliquots of 50-70 ml increasing to 150ml. To ensure adequate lung clearance lavage was done in both supine and prone position and physiotherapy was given during procedure. A total of 6000 ml of saline was instilled and 6600 ml was drained out in the procedure which lasted 6 hours.

The child was hemodynamically stable and maintained saturation through-out the procedure. Patient was gradually weaned off from ECMO and extubated on the subsequent day in the PICU.



Fig 3. Gradual clearing of fluid from proteinaceous material

There was excellent response both clinically and radiologically within 3-4 days of the procedure.

Patient was discharged and is off oxygen support. FU at 1 year revealed a well child with a weight gain of 3 kg.

Case 2:

3 year 1 month old child born of non consanguineous marriage presented to our hospital with complaints of cough, shortness of breath and progressive hypoxemia since the age of 8 months. She was on home oxygen of 4 litres through nasal prongs since 1 month. On examination she had severe failure to thrive with a weight 7 kg. Her vitals revealed a HR of Fig. 4. Chest X-ray after whole lung 140/minute, RR of 32-35/min. SPO2 was 96% on 4 litres of oxygen. On systemic examination child was having subcostal and intercostal retraction with bilateral coarse crackles on auscultation. Other systems were normal

CT chest was suggestive of diffuse ground glass and reticular densities in both lungs s/o PAP.

NGS showed a CSF2R mutation involving Exon 5-14 homozygous s/o pulmonary surfactant metabolism dysfunction type 4 hence confirming a diagnosis of Congenital pulmonary alveolar proteinosis. In view of progressive hypoxemia whole lung lavage was tried twice at another centre with single lung ventilation, but the procedure was abandoned midway as the child became hypoxic. Based on our earlier experience a decision to do whole lung lavage on ECMO and mechanical ventilation

was made. The difference in this patient was the use of two cannulas instead of one ECMO cannula. An 8 F ECMO cannula was inserted in right femoral vein for access and 12 F cannula was inserted to right IJV for return of blood. Tip of return cannula was positioned in right atrium with the help of $\mathbf C$ arm. This reduced the cost of the procedure significantly. ECMO flow and sweep gas flow were titrated to optimize oxygenation and ventilation. Lung lavage was done, one lung at a time with 50 ml aliquots of prewarmed saline while ventilating other lung with 3.5 F cuffed tube to optimize ventilation and oxygenation. Position change and physiotherapy was done during the total lung lavage to optimise the efficacy. Total lung lavage was continued till the returning fluid became less milky and clear. The child was weaned of ECMO in 2 hours post procedure and extubated the subsequent day. The child improved clinically and required oxygen in decreasing concentration over the nest 15 days. Follow up after a month revealed a weight gain of 500gms and decreased oxygen requirement to 1 litre/min only during sleep.

The majority of cases of hereditary PAP due to CSF2RA abnormalities developed progressive dyspnoea, exercise intolerance, tachypnoea and hypoxemia at median age of 3.5 years with failure to thrive reported in 55% patients. The



lavage with marked radiological improvement



Fig 6. Chest x ray showed widespread bilateral reticular shadowing air space disease (Pre-procedure)

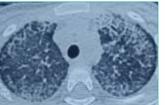


Fig. 5. Computed tomographic scan of the chest after whole lung lavage with significant radiological improvement



Fig. 7: Computed tomographic scan of the chest showed ground glass opacification and interlobular septal thickening (crazy paving pattern) (a) Upper lobe (b) Lower lobe



Fig. 8: CXR with ECMO cannula in

standard palliative treatment for hereditary PAP due to CSF2RA gene mutation is whole lung lavage. In small children, this method has technical difficulty because of the small size of trachea and unavailability of correct size of double lumen endotracheal tube which can lead to difficulty in ventilation during the procedure. There are some case reports in adults and very few case report in children where ECMO or cardiopulmonary bypass has been used for optimising oxygenation and ventilation for doing total lung lavage. 345 The advantage of ECMO being adequacy of oxygenation during the entire procedure. 6

REFERENCES:

- Trapnell BC, Whitsett JA, Nakata K. Pulmonary alveolar proteinosis. New England Journal of Medicine. 2003 Dec 25;349(26):2527-39.
- Martinez-Moczygemba M, Doan ML, Elidemir O, Fan LL, Cheung SW, Lei JT, Moore JP, Tavana G, Lewis LR, Zhu Y, Muzny DM. Pulmonary alveolar proteinosis caused by deletion of the GM-CSFRa gene in the X chromosome pseudoautosomal region 1. The Journal of experimental medicine. 2008 Nov 24.205(12).2711-6
- Seard C, Wasserman K, Benfield JR, Cleveland RJ, Costley DO, Heimlich EM. Simultaneous bilateral lung lavage (alveolar washing) using partial cardiopulmonary bypass: report of two cases in siblings. American Review of Respiratory Disease. 1970 Jun;101(6):877-84.
- Badiozaman R, Tahereh P, Shideh D, et al. . Whole lung lavage of nine children with pulmonary alveolar proteinosis: experience in a tertiary lung center. 4. Iran J Pediatr 2013; 23: 95-99
- Sihoe AD, Ng VM, Liu RW, Cheng LC. Pulmonary alveolar proteinosis in extremis: the case for aggressive whole lung lavage with extracorporeal 5. membrane oxygenation support. Heart, Lung and Circulation. 2008 Feb 1;17(1):69-72.
- Hiratzka LF, Swan DM, Rose EF, Ahrens RC. Bilateral simultaneous lung lavage utilizing membrane oxygenator for pulmonary alveolar proteinosis in an 8-month-old infant. The Annals of Thoracic Surgery. 1983 Mar 1;35(3):313-7.

LILAVATI HOSPITAL



MOLECULAR TESTING LABORATORY



Exceptional Specificity & Accuracy



Timely Diagnosis



Effective Personalized Treatment

BETTER MEDICAL OUTCOMES IN AREAS OF



Disease



Sexual & Reproductive Health



Management



Oncology Transplant

GET RTPCR COVID - 19 SWAB TEST RESULT ON THE SAME DAY

CASE REPORT V: ORTHOPAEDICS

The Black Knee - A Case Report

Dr. Amyn M. Rajani, MS Orth, D Orth, MBBS, Consultant - Orthopedics

What to Learn from this Article?

Thorough arthroscopic evaluation can help diagnose a rare case of ochronotic arthropathy and alleviate symptoms, further avoiding or delaying the need of arthroplasty.

Conflicts of Interest: Nil. **Source of Support:** None

Abstract

Introduction:

Ochronotic arthropathy in patients with alkaptonuria is a rare hereditary disorder. The altered metabolism causes the homogentesic acid derivatives to deposit in various connective tissues causing characteristic pigmentation. Due to the close clinical resemblance to that of a degenerative disorder, diagnosis of ochronotic arthropathy usually occurs intraoperatively. We report arthroscopic findings of a 50 yrs female with ochronotic arthropathy.

Case Report:

A 50 yrs old woman came with complaints of pain and swelling in the left knee. Clinical examination and MRI findings were correlated to reveal a tear of lateral meniscus. On arthroscopic examination, the blackish pigmentation of the meniscus and the articular cartilage led to the diagnosis of ochronotic arthropathy.

Conclusion: Arthroscopy plays an important role in diagnosis and treatment of patients with ochronotic arthropathy. The characteristic arthroscopic finding may aid diagnosis even in patients who do not have other systemic manifestations. Timely arthroscopic intervention can help delay the disease progression.

Introduction

Alkaptonuria is a rare metabolic disorder usually with an autosomal recessive trait. There is altered metabolism of phenylalanine and tyrosine amino acids due to deficiency of homogentisic acid oxidase (HAO) ^[1]. This causes excessive accumulation of homogentisic acid which further oxidises to form benzoquinone acetic acid (BQA). The characteristic bluish-black pigmentation called "ochronosis" of various connective tissues is due to these deposits of HAO and BQA ^[2]. Due to high affinity for fibrillary collagen, this pigment affects the hyaline cartilage of the larger peripheral joints. This results in decreased cross-linkage of collagen, thereby making it more susceptible to mechanical stress ^[3]. This causes the cartilage to become brittle, fragmented which loads the subchondral bone leading to inflamed synovium thereby resulting in "ochronotic arthropathy" ^[4].

Patients are usually asymptomatic till the fourth decade of life after which there is clinical presentation of ochronotic arthropathy ^[5]. Clinically, there is degenerative arthropathy of spine and the larger joints, lower limb being more common. The diagnosis is confirmed with histopathologic examination of tissue biopsies and assessment of homogentisic acid in the urine ^[6].

A 50 yrs old woman presented to us with a torn lateral meniscus. On arthroscopic examination, we found brownish-black pigmentation of the menisci and articular cartilage. With the suspect of alkaptonuria, further laboratory examinations of the urine sample and histopathologic examination of tissue biopsies were conducted which confirmed the diagnosis.

Case Report

A 50 yrs old woman came with chief complaints of pain and swelling in the left knee. She had a recent history of twisting injury while walking. Physical examination revealed tenderness along the lateral joint line and McMurray's test was positive for lateral meniscus. Flexion was terminally restricted and painful. Radiographs showed evidence of lateral compartment osteoarthritic changes. Tear of lateral meniscus (Fig. 1) along with synovitis (Fig. 2) and degenerative changes in the cartilage were evident on magnetic resonance imaging (MRI) of the left knee. Arthroscopic partial meniscectomy was planned on the basis of her clinical and radiological presentation.

Arthroscopic findings revealed hypertrophied and pigmented synovium (Fig. 3). There were small chondral defects of size 3 mm to 5 mm, Grade 2+ more over the lateral condyle than the medial condyle. There were pigmented loose bodies with size varying from 0.5 mm to 5 mm. Grade 3 changes were observed in the cartilage with kissing lesions and greyish-blackish discoloration. Both the menisci were discolored with tear in the lateral meniscus (Fig. 4). Although the integrity of all the ligaments was maintained, there was evident discoloration and thinning out.

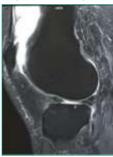
Partial lateral meniscectomy was performed with removal of loose bodies. Arthroscopic debridement was done to remove the hypertrophied synovium and specimen was sent for histopathological examination. With the suspect of ochronotic arthropathy due to the discoloration observed in arthroscopic intervention, further examination and investigation were planned.



Urine was normal in color but turned dark brown when tested with sodium hydroxide (NaOH). Urine showed high levels of HGA. Histopathological reports revealed brownish pigmentation along with degenerative changes. Detailed examination of the skin, sclera, eye, and ear showed no abnormalities.

Patient achieved immediate pain relief after the arthroscopic intervention. Postoperatively, physical therapy rehabilitation program, emphasizing on full weight- bearing walking, was started along with achieving pain-free range of motion followed by strength training. Patient had no complaints in the subsequent follow-ups at week 2, week 4, and week 8.







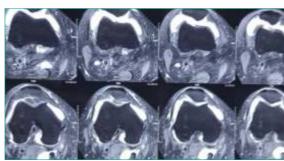


Fig 1. Miniscus Tear

Fig 2. MRI

Fig 3. Synovium

Fig 4. Effusion

Discussion

Ochronotic arthropathy is a rare disorder found in patients with alkaptonuria. Alkaptonuria is a metabolic disease which occurs in about 1 per billion births [7]. Albert and Zdareck studied the association between alkaptonuria and ochronosis in 1902 [8]. Few case reports of arthroscopic diagnosis and management of ochronosis have been reported. At present, no cure has been found for ochronosis or alkaptonuria. The rare prevalence of the disease makes it difficult to form a consensus on surgical treatment options.

The literature highlights the arthroscopic findings of ochronosis observed in knee and some in hip and shoulder [9,10,11]. The arthroscopic findings studied in this case report were consistent with the other cases documented. Case reported by Thacker et al. [11] described the arthroscopic findings in a 40 yrs old male with multiple joint involvements. The findings revealed pigmented hypertrophic synovium with loose bodies in the knee joint and frying of rotator cuff along with hypertrophic synovium in the shoulder joint. The patient did exhibit greyish-blackish discoloration on the surface, unlike in our case, where there was no obvious finding on the surface. The findings of pigmented hypertrophic synovium with loose bodies and lack of involvement of ligamentous structures, though less pliable than normal, were similar in our case. The involvement of the lateral meniscus in our case was consistent with the findings reported by Chen et al. [12], and Raaijmaaker et al. [13] where the patient had degenerative tear of menisci for which partial meniscectomy was performed. Generalized cartilage degeneration found in our case has also been reported consistently in the literature.

Arthroscopy in ochronotic arthropathy is recommended for diagnostic and therapeutic purpose. It helps to reduce pain and swelling and also improves range of motion. Removal of loose bodies and debridement of the frayed tissue can further reduce the insult to the articular cartilage. This helps in slowing down the rate of cartilage degradation, thereby delaying the need for arthroplasty. Therapeutic arthroscopy can thus provide immediate symptomatic relief and help impede the disease progression.

Conclusion

Arthroscopy as diagnostic and therapeutic tool plays an important role in the management of patients with ochronosis. The characteristic findings in this case report may help in diagnosis of ochronotic arthropathy without other systemic presentation. Arthroscopy can provide excellent symptomatic relief and avoid or delay the need for arthroplasty.

Clinical Message

Arthroscopy plays a vital role in diagnosis of ochronotic arthropathy as radiological findings including MRI may mimic degenerative or other traumatic conditions.

REFERENCES:

- 1. Mistry JB, Bukhari M, Taylor AM. Alkaptonuria. Rare Dis 2013;1:e27475.
- 2. Phornphutkul C, Introne WJ, Perry MB, Bernardini I, Murphey MD, Fitzpatrick DL, et al. Natural history of alkaptonuria. N Engl J Med 2002;347:2111-21.
- 3. Taylor AM, Boyde A, Wilson PJ, Jarvis JC, Davidson JS, Hunt JA, et al. The role of calcified cartilage and subchondral bone in the initiation and progression of ochronotic arthropathy in alkaptonuria. Arthritis Rheum 2011;63:3887-96.
- 4. Hamdi N, Cooke TD, Hassan B. Ochronotic arthropathy: Case report and review of the literature. Int Orthop 1999;23:122-5.
- 5. Cetinus E, Cever I, Kural C, Ertürk H, Akyıldız M. Ochronotic arthritis: Case reports and review of the literature. Rheumatol Int 2005;25:465-8.
- $6. \qquad \text{Millea TP, Segal LS, Liss RG, Stauffer ES. Spine fracture in ochronosis. Report of a case. Clin Orthop Relat Res 1992; 281:208-11.}$
- 7. Garrod AE. The incidence of alkaptonuria: A study in chemical individuality. 1902. Mol Med 1996;2:274-82.
- 8. Zatkova A. An update on molecular genetics of Alkaptonuria (AKU). J Inherit Metab Dis 2011;34:1127-36.
- 9. Chen AL, Rose DJ, Desai P. Arthroscopic diagnosis and management of ochronotic arthropathy of the knee. Arthroscopy 2001;17:869-73.
- 10. Thacker M, Garude S, Puri A. Ochronotic arthropathy: Arthroscopic findings in the shoulder and the knee. Arthroscopy 2003;19:E14-7.

CASE REPORT VI: PAEDIATRIC SURGERY

A Novel Missense Mutation in a case of synchronous malignancy in a child: Is it Li-Fraumeni Syndrome?

Dr. Shruti Tewari, 6th year DNB Resident, Paediatric Surgery

Dr. Rahul Deo Sharma, 4th year DNB Resident, Paediatric Surgery

Dr. Sushma Achugatla, 3rd year DNB Resident, Paediatric Surgery

Dr. Surendra Singh, 1st year DNB Resident, Paediatric Surgery

Dr. Swati Kanakia, MD, DCH, PhD, Consultant - Paediatric Hemato-Oncologist

Dr. Rajeev Redkar, M.Ch.(Paed. Surg), FRCS, DNB, MS (General Surgery),

FCPS, IAS, Consultant - Paediatric Surgery

Introduction

Synchronous malignancies are rare events in children. They pose a significant challenge in terms of chemotherapy and surgery^[1]. If there is an association with TP53, this challenge is intensified as the patients have a predisposition for development of tumors when exposed to chemotherapy or radiotherapy ^[2]. Li-Fraumeni syndrome (LFS) is a familial cancer syndrome associated with multiple malignancies and TP53 gene mutation ^[3]. We are reporting a case of a young child with multiple high-risk malignancies which are otherwise treated by high-dose chemotherapy and/or radiotherapy, but because associated with TP53 and MLH3 gene mutations, management strategy needed to be changed. The rarity of the gene mutations associated makes it a unique case and makes it close to being labeled as LFS.

Case Report

11 months old male child was admitted with the complaints of hoarseness of voice, generalized coarse hair overgrowth and hirsuitism from 8 to 9 months of age. An endocrine consultation was done for above symptoms followed by hormonal assay and radiological imaging studies.

High levels of serum cortisol (20.85 $\mu g/dL$), elevated testosterone levels (free testosterone 250 ng/dL and dihydrotestosterone 2500 ng/dL) were reported. Urinary Vanillyl-mandelic acid levels were normal but alpha-fetoprotein level were raised to 912 ng/mL (normal <10 ng/mL).

On computed tomography (CT), a heterogeneous and well encapsulated mass was reported in the left adrenal gland, with areas of focal calcification within the tumor [Figure 1a]. Simultaneously, another exophytic mass was visualized in the segment VI of the liver without any extension outside liver or within inferior vena cava (IVC), portal vein, or any nodal involvement [Figure 1b]. CT also reported a well-encapsulated mass extending between T4 and T7 vertebrae in the posterior mediastinum without any intra-spinal extensions [Figure 1c]. The lungs as well as the brain were clear of metastasis.

The child did not have any dysmorphic features. There was no history of malignancy in the siblings. The mother had a history of six previous abortions and hormonal treatment during pregnancy. The abortions were not spontaneous but for wanting a male child. The child's father had been formerly treated for renal cell carcinoma and had undergone partial nephrectomy 5 years ago.

Since three separate masses were detected in this child, we decided to biopsy one of them to identify the nature of the mass and the safest option was to perform a liver biopsy. An ultrasound-guided liver biopsy was done under corticosteroid cover, and it was reported as fetal variant hepatoblastoma (ICD-O-3 code 8970). Following that, a right thoracoscopic biopsy was done to rule out metastasis, but surprisingly it was reported to be a neuroblastoma stroma poor type (ICD-O-3 code 9490).

After ruling out adrenal medullary involvement (by urinary Vanillyl mandelic acid and homovanillic acid level) to prevent the intraoperative adrenal storm, the patient underwent a left adrenalectomy [Figure 2a] as well as excision of hepatoblastoma under corticosteroid cover [Figure 2b]. On definite histopathology report, the left adrenal mass was reported as adrenocortical malignancy (ICD-O-3 code 8370) without a breach in the capsule and the hepatoblastoma was of fetal variant without any hepatic vein, IVC, or extra-hepatic involvement and clear margins of resection. We excised the chest mass 2 weeks later and it was reported as well differentiated, stroma poor type neuroblastoma [Figure 2c].

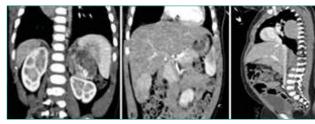


Figure 1: (a) Radiological image of adrenocortical carcinoma, (b) radiological image of hepatoblastoma, (c) radiological image of neuroblastoma

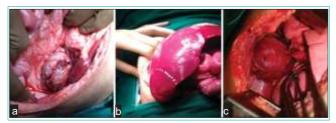


Figure 2: (a) Intraoperative picture of adrenocortical carcinoma, (b) intraoperative picture of hepatoblastoma, (c) intraoperative picture of neuroblastoma



Post tumor excision, the hormonal levels of cortisol (2.0 μ g/dL), testosterone (free testosterone 0.1 ng/dL and dihydrotestosterone 27 ng/dL), and alpha-fetoprotein (3.0 ng/mL) were reduced. The steroids were eventually stopped after tapering doses.

Gene mutation studies were done as the child presented with three histologically distinct malignancies and it revealed a germline TP53 gene mutation (c. C530G, p. Pro177Arg) with allelic ID 468390 and MLH3 gene (c. G2221T, p. Val741Phe) mutations. NMYC amplification was confirmed on Interphase fluorescent in situ hybridization (FISH) technique which revealed a 15-fold increase of NMYC signals to chromosome enumeration probe signals (Table 1). Following this, multiplex ligation-dependent probe amplification (MLPA) assay was also performed on the neuroblastoma tissue to check for segmental chromosomal anomalies on chromosomes 1, 2, 3, 4, 7, 9, 11, 12, and 17 [Table 1]. The parents were offered to undergo genetic testing to find out any genetic abnormalities, but they refused to undergo such tests.

Genetic	Copy status	Significance
alteration		
ALK	Normal	Helps activating NMYC, high-risk tumor
DDX1	Amplification	High-risk tumor
NMYC	Amplification	High-risk tumor
1p	None	Near-diploid or tetraploid tumors
11q23	Normal	Higher disease state and poor prognosis
17q	Gains	Poor outcome
2p	Amplification	Very aggressive course
<i>3p</i>	Normal	Tumor suppressor gene, higher age at diagnosis
4p	Normal	Tumor suppressor gene
7 <i>q</i>	Normal	Tumor suppressor gene

Table 1: Genetic and molecular findings on multiplex ligation-dependent probe amplification and fluorescent in situ hybridization in cases of neuroblastoma and its significance

While on cisplatin and doxorubicin regimen chemotherapy (drugs common for both hepatoblastoma and neuroblastoma), patient presented with convulsions (probably hypoglycemic) and shock requiring inotropic support and cardiopulmonary resuscitation. He was eventually diagnosed to have severe chemoport-site sepsis (culture positive), which had to be removed once stable. Post this incident, the child developed hypoxic brain injury with neurological deficits in the form of weakness and regression of milestones. He is now on the road to recovery. At the time of reporting this case, the child is not on any chemotherapy and undergoing limb physiotherapy.

Discussion

Malignancies with different histopathological origin in one person are termed as primary multiple tumors. The incidence has increased incidence nowadays due to early detection by better diagnostic techniques [4]. TP53 mutation and LFS are often reported with multiple malignancies and are most commonly associated with adrenocortical malignancies, soft-tissue sarcomas, brain tumors, and breast tumors [5].

Multiple criteria have been laid down for the identification of LFS and Li-Fraumeni-like syndrome (LFL) like Birch and Eeles criteria [6]. Another syndrome known as hereditary neoplastic syndrome is also associated with multiple malignancies and is characterized by tumor occurrence at an early age. However, that is associated with endocrine tumors [7].

Our patient does not fit into any criteria to be labeled as LFS or LFL, even with the presence of a renal cell carcinoma in his father. This makes it a unique presentation with germline TP53 mutation and three histologically different malignancies. This is a novel mutation, but its clinical significance needs to be proven in future.

The age of presentation is also unique as Synchronous malignancies are most commonly seen in the second or third decade [8]. Thus this is another unique aspect of our case as three distinct malignancies are reported at the age of 11 months. Three other reports were found where the patients are aged 6, 8, and 10 months [3,9,10].

There are multiple case reports of neuroblastoma reported with TP53 gene mutation while there are six reported cases of TP53 mutation with adrenocortical cancer (ACC) and neuroblastoma. This case is seventh such report [Table 2].

The reported mutation in TP53 gene in our present case is a missense mutation c. C530G, p. Pro177Arg (P177A) with allelic ID 468390. This germline mutation presents within the DNA-binding domain of TP53 but its effect on TP53 protein function is still biochemically uncharacterized [13]. According to the International Agency for Research on Cancer (IARC) TP53 database, there has been only one somatic TP53 P177A mutation identified in colorectal cancer and no germline TP53 P177A has been identified till August 2018. Totally, there are 498 gene variants identified with TP53 mutation in the IARC TP53 database [14].

The clinical exome sequencing also identified a mutation in the MLH3 gene which was a missense mutation c. G2221T,pVal741Phe (V741P) which is associated with hereditary non-polyposis colorectal cancer (HNPCC) type $7^{[15]}$. According to the Centre for Genomic Study and Genetics, Zhejiang University, there are a total of 54 gene variants which are described for MLH3 mutation and most of them are on exon 2, as in our case. According to the same genetic registry, only two other cases have been reported with V741P mutation, one in an adult familial endometrial carcinoma and another in a case of colorectal cancer [16].

This case is did not fit into any criteria laid down for LFS in the presence of TP53 mutation and ACC with neuroblastoma. The reported gene mutation is a novel missense mutation which has never been reported in germline mutation, while only one case is reported that of a somatic mutation (with colorectal cancer) [14]. The MLH3 gene mutation variant V741P has not been previously associated with any of the malignancies seen in this child [16]. This indirectly may point to the fact the child bears a risk of developing future malignancy due to this genetic aberration.

The MLPA assay and NMYC amplification makes the neuroblastoma a high-risk tumor. The treatment regimen for such cases consists of high-dose chemotherapy, irradiation, followed by immunotherapy [17]. This presents a unique dilemma for the treating doctors for planning further course of treatment as radiotherapy or high-dose chemotherapy cannot be administered because of TP53 mutation. This specific mutation predisposes the patients for development of more malignancies when exposed to radiation or other cytotoxic drugs.

A management protocol for these patients with multiple malignancies cannot be formulated as these are one-off presentation and each such presentation must be dealt individually.

Case	Location	Year	Age/sex	Function	Gene variant	Exon	Other associated	N-MYC	Reference
number							malignancy	amplification	
1	The USA	1998	18 months/male	Loss of function	R248W 45XO	7	ACC	No	[1]
2	The USA	2008	10 months/male	Loss of function	R248W	7	ACC	No	[10]
3	Brazil	2015	NA	Loss of function	R337H	10	ACC	Present	[11]
4	Brazil	2015	NA	Loss of function	R337H	10	ACC	Present	[11]
5	The USA	2015	8 months/male	Unknown	I162F	5	ACC	No	[9]
6	China	2017	6 months/male	Loss of function	N268E	8	ACC	No	[3]
7	The UK	2014	2 years/male	Loss of function	R248Q	Whole exome	Benign myofibroblastic	Present	[5]
							proliferation and sarcoma		
8	The USA	2013	3 years/male	Loss of function	P219S	Whole exome	High-risk neuroblastoma	Present	[2]
9	The UK	1992	1 year/male	Loss of function	R273H	NA	Osteosarcoma	NA	[12]
10	India	2018	11 months/male	Unknown	R177A	5	Hepatoblastoma and ACC	Present	This study

Table 2: Cases of neuroblastoma combined with other tumors and TP53 mutation

Conclusion - Take-Home Message

Multiple synchronous malignancies are a rare occurrence in children. Cytogenetic (karyotyping, FISH, and comparative genomic hybridization) and molecular cytogenetic (MLPA and restriction fragment length polymorphism) studies help us to identify the known mutations, whereas other molecular cytogenetic techniques (denaturing gradient gel electrophoresis, single-strand conformational polymorphism, and heteroduplex analysis) help to identify the unknown mutations. Genetic mapping of parents and siblings is crucial to identify inherited mutations or de novo mutations. The treatment protocol has to be tailor made for these patients and requires a multidisciplinary approach. Regular follow-up with an eye out for a possible malignancy is of utmost importance.

REFERENCES:

- 1. Pivnick EK, Furman WL, Velagaleti GV, Jenkins JJ, Chase NA, Ribeiro RC. Simultaneous adrenocortical carcinoma and ganglioneuroblastoma in a child with Turner syndrome and germline p53 mutation. J Med Genet 1998;35:328-32.
- 2. Pugh TJ, Morozova O, Attiyeh EF, Asgharzadeh S, Wei JS, Auclair D, et al. The genetic landscape of high-risk neuroblastoma. Nat Genet 2013;45:279-84.
- 3. Tang YJ, Yu TT, Ma J, Zhou Y, Xu M, Gao YJ. Composite adrenocortical carcinoma and neuroblastoma in an infant with a TP53 germline mutation: A case report and literature review. J Pediatr Hematol Oncol 2019;41:399-401.
- 4. Moertel CG, Dockerty MB, Baggenstoss AH. Multiple primary malignant neoplasms. I. Introduction and presentation of data. Cancer 1961;14:221-30.
- 5. Behjati S, Maschietto M, Williams RD, Side L, Hubank M, West R, et al. A pathogenic mosaic TP53 mutation in two germ layers detected by next generation sequencing. PLoS One 2014;9:e96531.
- 6. Tinat J, Bougeard G, Baert-Desurmont S, Vasseur S, Martin C, Bouvignies E, et al. 2009 version of the Chompret criteria for Li Fraumeni syndrome. J Clin Oncol 2009:27:e108-9.
- 7. Frank TS. Hereditary cancer syndromes. Arch Pathol Lab Med 2001;125:85-90.
- 8. Gonzalez KD, Noltner KA, Buzin CH, Gu D, Wen-Fong CY, Nguyen VQ, et al. Beyond Li-Fraumeni syndrome: Clinical characteristics of families with p53 germline mutations. J Clin Oncol 2009;27:1250-6.
- 9. Courtney R, Ranganathan S. Simultaneous adrenocortical carcinoma and neuroblastoma in an infant with a novel germline p53 mutation. J Pediatr Hematol Oncol 2015;37:215-8.
- 10. Rossbach HC, Baschinsky D, Wynn T, Obzut D, Sutcliffe M, Tebbi C. Composite adrenal anaplastic neuroblastoma and virilizing adrenocortical tumor with germline TP53 R248W mutation. Pediatr Blood Cancer 2008;50:681-3.
- Seidinger AL, Fortes FP, Mastellaro MJ, Cardinalli IA, Zambaldi LG, Aguiar SS, et al. Occurrence of neuroblastoma among TP53 p.R337H Carriers. PLoS One 2015;10:e0140356.
- 12. Porter DE, Holden ST, Steel CM, Cohen BB, Wallace MR, Reid R. A significant proportion of patients with osteosarcoma may belong to Li-Fraumeni cancer families. J Bone Joint Surg Br 1992;74:883-6.
- 13. Kotler E, Shani O, Goldfeld G, Lotan-Pompan M, Tarcic O, Gershoni A, et al. A systematic p53 mutation library links differential functional impact to cancer mutation pattern and evolutionary conservation. Mol Cell 2018;71:178-90, e8.
- 14. IARC TP53 Search. Available from: http://p53.iarc.fr/TP53GeneVariations.aspx. [Last accessed on 2019 Jul 18].
- 15. Liu HX, Zhou XL, Liu T, Werelius B, Lindmark G, Dahl N, et al. The role of hMLH3 in familial colorectal cancer. Cancer Res 2003;63:1894-9.
- 16. All genes-Zhejiang University Center for Genetic and Genomic Medicine (ZJU-CGGM) & Dian Diagnostics. Available from: http://www.genomed.zju.edu.cn/LOVD3/genes [Last accessed on 2019 Jul 18].
- 17. Casey DL, Kushner BH, Cheung NK, Modak S, LaQuaglia MP, et al. Local control with 21-Gy radiation therapy for high-risk neuroblastoma. Int J Radiat Oncol Biol Phys 2016;96:393-400.

FIND THE BEST TREATMENT PLAN FROM THE BEST PLASTIC SURGEONS



CASE REPORT VII: PLASTIC SURGERY

Locally Advanced Rectal Carcinoma: A reconstructive challenge

Pratik Vijay Shah, Fellow (Microsurgery) Archana Nehe, Post Graduate, General Surgery Leena Jain, Consultant Plastic and Microvascular Surgeon Samir Kumta, Consultant Plastic and Microvascular Surgeon Deepak Chabbra, Consultant Oncosurgeon

A, 45 years old lady presented with complaints of growth in the perianal area, increasing in size over last one year; increasingly associated with discomfort now due to which she sought treatment. She gave history of radiation therapy about two years ago for proven rectal cancer, however was unwilling for advised surgical resection and was lost to follow-up.

Examination revealed a locally advanced ulceroproliferative rectal cancer measuring about 12 x 10 cms in the perianal and perineum region with surrounding induration. Distal part of posterior vaginal wall was indurated, however, cervix was free (figure 1). There were no metastatic lesions.

She was planned for a laproscopic abdomino-perineal resection, end colostomy and reconstruction. Laproscopic abdominal resection was done to reduce morbidity of a laprotomy and an end colostomy was done in left paramedian area. The perineal resection was then proceeded with, with removal of posterior vaginal wall; the final defect measured 20 X 15cms (figure 2).

Reconstruction was planned on the following principles:

- 1. Separation of abdominal and pelvic cavities to prevent any internal herniation
- 2. Reconstruction of posterior vaginal wall to restore her sexual function
- 3. Reconstruction of perineal and perianal tissue with a robust cover
 - a. To fill the dead space with vascularised tissue to prevent seroma, infection, etc.
 - b. To enable normal movements without any limitations
 - c. To sustain radiation

Conventional reconstruction plan: usually for such defects, inferiorly based, rectus abdominis myocutaneous flap through a laprotomy approach. The resection was planned laproscopically to reduce morbidity of an open abdominal surgery. The rectus flap provides muscle to be used as a filler and soft tissue for cover in one go. However, it weakens the abdominal wall infraumbilically and needs a mesh to reconstruct the abdominal wall. The abdominal weakness is compounded due to placement of stoma on the other side of midline.

Modified Reconstruction plan (figure 3):

- 1. Biological mesh to separate the abdominal and pelvic cavities fixed to the pelvic inlet.
- 2. Pedicled anterolateral myocutaneous flap to provide muscle to fill in the cavity and skin cover to resurface the defect (figure 4 and 5).
- Pedicled gracilis muscle flap to reconstruct the posterior vaginal wall (figure 6).
- 4. Inset of flaps to recreate the introitus.

The modified plan, enabled us to harvest two flaps from a single thigh that could be used independently to reconstruct different constituents of the defect created after tumor excision. Donor site morbidity was negligible. Her post-operative period was uneventful. Her three month imaging studies showed no evidence of recurrence.

With the wide arc of rotation, pedicled perforator flaps have numerous applications while significantly reducing donor site morbidity. Perforator flaps are now first choice for reconstruction of complex defects and a combination of flaps enable one to provide a three dimensional construct.



Figure 1: Ulceroproliferative rectal growth with induration tumor excision with loss of in the perianal region



Figure 2: Defect after posterior vaginal wall



Figure 3: Harvest of gracilis and anterolateral thigh myocutaneous flap from left thigh



Figure 4: Wound healed with reconstruction of



Figure 5: Well settled flap



Figure 6: Posterior vaginal wall formed by gracilis flap as shown in post-operative ultrasound





Call: 8291280428

between 8am to 6pm Monday to Saturday (excluding Sundays and Public Holidays)

List of Publications (International)

S. No.	Author	Title of the Paper / Chapter	Name of Journal
1	Dr. Ajit Menon,	Approach to Asymptomatic Case of	Journal of Clinical Cardiology and
	Dr. Parag Bhalgat,	Bicuspid Aortic Valve with	Cardiovascular Interventions
	Dr. K.V Charan Reddy,	Coarctation and Massive Aortic	Auctores Publishing – Volume
	Dr. Abhishek Shah,	Arteriopathy: a Ticking Time Bomb	4(10)-169 www.auctoresonline.org
	Dr. Sanjeev Vichare		ISSN: 2641-0419

List of Publications (National)

S. No.	Author	Title of the Paper / Chapter	Month of Publication
1	Dr. Rajeev Redkar,	Comparison between Suction Rectal	Journal of Indian Association of
	Dr. Swathi Chigicherla,	Bipsy and Full –Thickness Rectal	Pediatric Surgeons, 2021;26:144-7.
	Dr. Shruti Tiwari,	Biopsy in the Diagnosis of	
	Dr. Rahul Deo Sharma	Hirschsprung's Disease	
2	Dr. Vasant Nagvekar,	Clinical Outcome of Patients on	Indian J Crit Care Med
	Dr Anand Shah,	Ceftazidime-avibactam and	2021;25(7):780-784.
	Dr. Vrajesh Kumar,	Combination Therapy in	
	Dr. P Unadkat,	Carbapenem-resistant	
	Dr. Amol Chavan,	Enterobacteriaceae	
	Dr. Ruhi Kohli,		
	Dr. Shailendra Hodgar,		
	Dr. Aashita Ashpalia,		
	Dr. Niranjan Patil,		
	Dr. Rahul Kamble		



Straight from the Heart - Patient Testimonials

This is with reference to the post COVID-19 packages that Lilavati Hospital has come up with for the people who were diagnosed with the mentioned virus.

I was hesitant is spending the amount for the Basic care package, but the way you explained it to me about the advantages it just changed my mind. I sincerely appreciate you explaining the same thing to me 4 times when I called you up without any hesitation.

The blood tests, and other tests in the package were so swiftly arranged, I felt like I was treated like a VIP person.

Post-test consultation from the Dietician and the Pulmonologist was superb. The time they spent explaining the things was just outstanding.

I sincerely thank you and your team for the wonderful service that I received for my package.

PRATHAMESH BELGE

I liked the cleanliness of the Hospital and systematic procedure

GOPAL CHAVAN

Good staff, good & clean area, good environment. I am happy with Lilavati Hospital. Thank you!

SAMIKSHA TAMBEKAR

Nursing staff are very competent & interactive in services.

YATIN SAMANT

I like Health check-up staff & doctors & their behaviour, they took utmost care. Overall cleanliness is also good.

AMRISH SINHA

All the queries are handled really very good, it's worth coming here all the way from Pune.

VARSHA KHANDARE

This Hospital is with excellent services.

LATA AGARWAL

The staff & doctors are very co-operative. We can sway excellent services rendered by doctor & all Hospital team.

JAIPRAKASH ABICHANDANI

Lilavati Hospital is a reputed and best Hospital in Mumbai. All services are under one roof.

KAMRAN

The staff at every step of Health check-up are very helpful.

BRYNA MENEZES

I like the punctuality of all, systematic & comprehensive procedure for Periodic Medical Examination.

AJAY BHATNAGAR

Recently, I was admitted to Lilavati hospital for being a Covid-19 positive with some lung infection. I must admit I was slightly apprehensive to get admitted due to the bad media publicity about the private hospitals. But my general practitioner doctor insisted that I remain under expert medical supervision at Lilavati, and he couldn't be more correct.

Right from the admission and triage to the discharge process, I had a very positive and compassionate experience. The whole staff on the Covid-19 floor was very helpful despite being in a tough situation. Having to wear a PPE kit whole day and going through all the drills with the patients must be hard. Yet they never showed any fatigue or discontent and were equally cheerful and courteous right to the end of their shifts. I learnt from them that the staff worked 6 hours a shift and for the shifts that start or end at an odd time such as 2am, hospital has provided them with the beds and relaxation area on the fifth floor. This is really generous from the hospital management to look after their staff's safety and well-being in a best possible way.

I must also mention how good food and cleaning services staff was. Meals were always delivered on time and were nutritious yet tasty. Cleaning staff was diligent, every few hours they would clean the floor and apply disinfectant to the floor and other areas such as bathroom, sitting coach etc. Head nurse visited everyday to confirm everything is in the order. She even noticed I am using a same cloth mask for couple of days and got the services staff to dispense disposable masks not only to me but all the patients on the floor, to avoid any possibility of mucormycosis especially with the senior patients.

I initially had a lot of anxiety especially about my fluctuating saturated oxygen levels, possible need for much dreaded Remdesivir and further deterioration of the lung infection. However, RMOs and senior nursing staff assured me that everything is under control and will improve with the medications. They even encourage breathing exercises etc. to calm myself. My consulting doctor, Dr Pralhad Prabhudesai was expert in his advice and helped me get over the anxiety quickly. From the second day itself, he discussed about the possible discharge date. This was much promising.

On my last day, I offered to order some sweets and chocolates for the staff on the floor, purely as a token of appreciation. But this was quickly quashed by the nurses in the bay, saying it is their job and they don't expect anything. And they also said there are many behind the scenes and it would be unfair for only some of them to get any gift even if it is a token.

My sincere thanks to all the staff at Lilavati Hospital, particularly, Covid-19 carers and doctors on the 8th Floor. They are doing their personal best to overcome this tough situation and influx of many Covid-19 patients. I wish them all the best. May God bless them always.

VISHWAS APTE

I like the cleanliness, helpful and friendly nature of staff, doctors, nurses & housekeeping staff.

RITA RAIKAR

Prompt diagnosis of my condition and exceptional care from Nursing staff. Very compassionate & provided utmost care & support.

SHRUSTI POOJARY

Initial emergency care was prompt. The good nursing staff everywhere, ICU, Stroke Unit & ward.

DIXON MIRANDA

I like convenience, cleanliness & cordial staff at all levels.

SUDHIR GUPTA

Very homely atmosphere which helps in the quick improvement of the patient's health.

SAJINI BHATIA

The courtesy and willingness to help & take care of the patients. All are super friendly!

VIJAYA PANCHAL



Services Available

MEDICAL

Anesthesiology

Audiology and Speech Therapy

Cardiology Chest Medicine

Chronic Pain Management

Dental

Dermo Cosmetology

Diabetology & Endocrinology

Gastroenterology

Diagnostics & Therapeutic Endoscopy

Haematology Hair Transplant

Head and Migraine Clinic

Internal Medicine
Infectious Diseases

Lactation

Medical Oncology Chemotherapy Nephrology Neurology

Psychiatry / Psychology / Neuropsychology

Physiotherapy Pediatrics Rheumatology Sleep Medicine

SURGICAL

Bariatric Surgery Cardiothoracic Surgery Cochlear Implant Surgery Colorectal Surgery Diabetic Foot Surgery

Endocrine Surgery

ENT and Head & Neck Surgery

Gastro Intestinal Surgery

General Surgery

Gynecology, Obstetrics & IVF Minimal Invasive Surgery (Laproscopic Surgery)

Neuro Surgery Onco Surgery Ophthalmology

Orthopedics, Sports Medicine

Pediatric Surgery

Plastic & Reconstructive Surgery

Spine Surgery

Transplants: Heart, Corneal, Kidney &

Liver

Urology, Andrology Vascular Surgery

24 HRS IMAGING

T Scan

Interventional Radiology

MRI

Non Invasive Cardiology

CATH Lab Sonography X-Ray

CRITICAL CARE

Intensive Care Unit (ICU)

Intensive Cardiac Care Unit (ICCU)

Neo-Natal Intensive Care Unit (NICU) Paediatric Intensive Care Unit (PICU)

Paralysis & Stroke Unit

Surgical Intensive Care Unit (SICU)

DIAGNOSTIC

Audiometry EEG / EMG Health Check-up

BMD

Mammography Nuclear Medicine PET & SPECT CT Scan

Urodynamics

24 HRS LABORATORY SERVICES

Blood Bank Histopathology Microbiology Pathology

OTHER 24 HRS SERVICES

Ambulance Emergency Pharmacy Roshni Eye Bank

HYDROTHERAPY CENTRE

Benevolence

The social service wing of the hospital - SEWA serves to the health requirements of needy people. This department seeks to bridge the gap between the needy patients and the fast evolving medical technology. Various social activities such as free OPD, services to senior citizen, sending mobile vans to Adivasi areas to organize free health check-up camps, free camps are undertaken as an on-going process. The Roshni Eye Bank managed by Lilavati hospital is a well-equipped comprehensive centre for cornea removal, processing, storing, supplying and corneal transplantation.

Under this service Lilavati Hospital & Research Centre offers:

- 1. Free OPD
- 2. Health Check up Camps at Nana Nani Parks
- 3. Mobile Clinic
- 4. Roshni Eye Bank

BENEFICIARIES	for F.Y 2020-2021
Free OPD	14,382
Mobile Clinic	15,161

Important Telephone Numbers

Tall Face	10003/70/13
Toll Free	18002678612
Emergency / Casualty	8063 / 8064
Hospital Fax	+91 22 2640 7655
Ambulance	+91 9769250010
TPA Fax	+91 22 2640 5119
Boardline	+91 22 2656 8000 / +91 22 2675 1000
Extensions	
Admission Department	8080 / 8081 / 8082
AKD Counter	8650 / 8651
Appointment - OPD	8050 / 8051
Billing - Inpatient	1586
Billing - OPD	8052
Blood Bank	8215
Blood Bank Medical Social Worker	8214
Cardiology	8236
Cath Lab	8137
Chemist	1579 / 1578
CT Scan Department	8044
Dental	8020
Dermatology / Hydrotherapy	8021
EMG / EEG	8249 / 8250
Endoscopy	8057
ENT / Audiometry	8232
Health Check-up Department	8354 / 8356
IVF	8226
Medical Social Worker (SEWA)	8361
MRD	8358 / 8359
MRI Department	8066
Nuclear Medicine / PET & SPECT CT	8092
Ophthalmology	8229
Physiotherapy	1536
Report Dispatch Counter	1620
Sample Collection Room	8028
TPA Cell	8089
Transplant Co-ordinator	8362
Urodynamics	8032
Visa Section	8248 / 8244

8038

X-Ray, Sonography Department



Few Honorable Mentions





Infectious Diseases Society of America (IDSA) Board of Directors elected Dr. Vasant Nagvekar, Consultant – Infectious Diseases as an IDSA Fellow. Fellowship in the Society is recognition of, and honor conferred upon, those who have achieved professional excellence in the field of infectious diseases.



Dr. Khyati Dedhia , Consultant - Radiology & Dr.Mona Mehta, Consultant - Radiology won 2nd prize in poster category for their poster 'Covid-19 vaccine related axillary adenopathy- what radiologists should know, when to schedule mammography appointments' in an online national conference called- Breast Imaging Master class held on May 29-30, 2021 organized by Indian Radiologist.



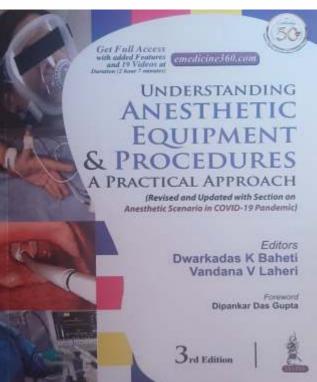


AVATAR (Association of Vascular Access & Interventional Renal Physicians) Foundation has conferred Guru Dronacharya award to Dr.Hemant Mehta , Consultant – Nephrology for his outstanding contribution, dedicated to education and promotion of Renal Science in India.



Dr Rajeev Redkar - Senior Consultant Paediatric Surgery has been elected as the Chairman of the Maharashtra Chapter of Indian Association of Paediatric Surgeon.

Dr Redkar has also been indicated as a member of the specialist board of Paediatric Surgery of the National Board of Examination.



Dr. Dwarkadas Baheti, Consultant – Pain Management released 3rd edition of this book Understanding Anesthetic Equipment & Procedures – A Practical Approach

Key Features of the book:

- The First Book having a Dedicated section to the management of COVID patients during anaesthesia and critical care situations.
- This Book is first of kind which combines knowledge of Anaesthesia Equipment's and Procedures.
- Only book have DVD of demonstration of Procedures done in Anaesthesia.
- One book for all include students, practising anaesthesiologists and teacher's.
- The description of equipment's and procedures is in simple language which students from vernacular medium will also understand and follow easily.
- Every equipment and procedure have been described where not only the principle behind its working can be understood, but it is possible to explain it in theory or practical examination by the student.

Doctors Associated with Lilavati Hospital

Andrology

Dr. Shah Rupin S.

Anaesthesiology

Dr. Baxi Vaibhavi

Dr. Budhakar Shashank

Dr. Gandhi Nisha

Dr. Gaiwal Sucheta

Dr. Gawankar Prakash

Dr. Kharwadkar Madhuri

Dr. Khatri Bhimsen

Dr. Kulkarni Satish K.

Dr. Mahajan Anjula

Dr. Mascarenhas Oswald

Dr. Kothari Namrata

Dr. Patil Prajakta

Dr. Shah Falguni

Dr. Waradkar Samidha

Audiology & Speech Therapy

Mr. Bhan Satyan

Ms. Gorawara Pooja

Ms. Parulkar Bakul

Ms. Satam Sneha

Bariatric Surgery

Dr. Palep Jaydeep

Dr. Shah Shashank

Blood Bank

Dr. Saraswat Shubhangi

Cardiovascular & Thoracic Surgery

Dr. Bhamre Bipeenchandra

Dr. Bhanushali Amol

Dr. Bhattacharya S.

Dr. Chaudhri Babar

Dr. Honnekeri Sandeep T.

Dr. Irniraya Krishna Prasad

Dr. Jaiswal O. H.

Dr. Joshi Suresh

Dr. Kumar Pavan

Dr. Mehra Arun P.

Dr. Nand Kumar

Dr. Pandey Kaushal

Dr. Rachmale G. N.

Dr. Ravishankar V.

Dr. Vichare Sanjeev

Cardiology

Dr. Bajaj Harish

Dr. Ballani Prakash

Dr. Bang Vijay

Dr. Dargad Ramesh R.

Dr. Gokhale Nitin S.

Dr. Jhala Darshan

Dr. Kothari Snehal N.

Dr. Lokhandwala Yash

Dr. Mehan Vivek

Dr. Merchant S. A.

Dr. Menon Ajit R. Dr. Mehta Haresh G.

Dr. Nabar Ashish

Dr. Pillai M. G.

Dr. Pinto Robin

Dr. Punjabi Ashok H.

Dr. Rao Anand

Dr. Rao Ravindra Singh

Dr. Ratnaparkhi Gajanan

Dr. Samuel K. Mathew

Dr. Sanzgiri P. S.

Dr. Shah Chetan

Dr. Sheth Siddharth

Dr. Suratkal Vidya

Dr. Vijan Suresh

Dr. Vvas Pradeep R.

Dr. Vora Amit

Dr. Vajifdar Bhavesh

Chest Medicine

Chest Wieulchie

Dr. Chhajed Prashant

Dr. Mahashur Abha

Dr. Mehta Sanjeev K.

Dr. Prabhudesai P. P.

Dr. Parkar Jalil D.

Dr. Rang Suresh V.

Colorectal Surgery

Dr. Chulani H. L.

Dentistry / Dental Surgery

Dr. Bhavsar Jaydeep P.

Dr. Deshpande Dilip

Dr. Gala Jigar

Dr. Joshi P. D.

Dr. Khatavkar Arun

Dr. Kamdar Rajesh J.

Dr. Parulkar Darshan

Dr. Samath Shyamcharan

Dr. Sanghvi Sameer

Dermatology

Dr. Goyal Nilesh

Dr. Mehta Nimesh

Dr. Oberai Chetan

Dr. Parasramani S. G.

Dr. Pillai Jisha

Diabetic Foot Surgery

Dr. Rege Tushar

Dr. Vaidya Sanjay

Diabetology

Dr. Panikar Vijay

Diabetology & Endocrinology

Dr. Joshi Shashank R.

Dr. Naik Vaishali **Dietician**

D. D.: I

Dr. Pai Veena

ENT

Dr. Dhingra Preeti

Dr. D'souza Chris E.

Dr. Jayashankar Narayan

Dr. Parasram Kamal S. Dr. Pusalkar A.

Endocrine Surgery

Dr. Agrawal Ritesh

Endo Urology

Dr. Utture Anand **Gastro Intestinal Surgery**

Dr. Bharucha Manoj

Dr. Kulkarni D. R.

Dr. Mehta Hitesh

Dr. Palep Jaydeep

Dr. Shaikh Taher

Dr. Varty Paresh

Dr. Wagle Prasad K.

Dr. Zaveri Jayesh P.

Foot and Ankle

Dr. Kini Abhishek

Gastroenterology

Dr. Barve Jayant S.

Dr. Choksi Mahul

Dr. Choksi Mehul

Dr. Kanakia Raju R.

Dr. Phadke Aniruddha Y.

Dr. Parikh Samir S.

Dr. Shah Saumil K.

General Surgery Dr. Garud T. V.

Dr. Mehta Narendra

Dr. Nikam Narendra

Dr. Trivedi Narendra

Gynaecology

Dr. Agarwal Rekha

Dr. Coelho Kiran S. Dr. Goyal Swarna

Dr. Medhekar Mansi

Dr. Nanavati Murari S.

Dr. Pai Hrishikesh

Dr. Pai Rishma D. Dr. Palshetkar Nandita

Di. I alsiletkai Ivai

Dr. Salunke Vivek Dr. Shah Cherry C.

Haematology

Dr. Agarwal M. B. Dr. Bhave Abhay

Hair Restoration

Dr. Agrawal Sumit

Dr. Nahar Raina

Headache & Migraine

Dr. Ravishankar K.

Healthcheckup Consultant

Dr. Desai Sandeep

HistopathologyDr. George Asha Mary

Dr. Tampi Chandralekha

Infectious Diseases Consultant Dr. Nagvekar Vasant C.

Intensivist / Physician
Dr. Jiandani Prakash

Dr. Kavita S.

Dr. Madkaikar Sneha

Dr. Shekade Kiran Dr. Shrinivasan R.

Dr. Vas Conrad Rui

Interventional Neuroradiology

Dr. Limaye Uday S. **Interventional Radiology**

Dr. Dharia Tejas

Dr. Rai Jathin Krishna Dr. Sheth Rahul

Dr. Warawdekar Girish



Joint Replacement Surgery

Dr. Maniar Rajesh N.

Lactation Consultants

Dr. Joshi Mugdha

Ms. Temkar Swati

Liver Transplant

Dr. Mehta Naimish

Dr. Shaikh Taher

Nephrology

Nephrology

Dr. Mehta Hemant J.

Dr. Shah Arun

Dr. Suratkal L. H.

Dr. Upadhyaya Kirti L.

Neurology

Dr. Chauhan Vinay

Dr. D'souza Cheryl

Dr. Deshpande Rajas

Dr. Sirsat Ashok M.

D G . G . 11

Dr. Soni Girishkumar

Dr. Vyas Ajay

Neuropsycology Ms. Panjwani Siddhika

Neuro Surgery

Dr. Ambekar Sudheer

Dr. Andar Uday

Dr. Dange Nitin

Dr. Goel Atul

Dr. Parekh Harshad

Dr. Pawar Sumeet

Dr. Ramani P. S.

Nuclear Medicine

Dr. Krishna B. A.

Dr. Shimpi Mahajan Madhuri

Oncology

Dr. Lokeshwar Nilesh

Dr. Menon Mohanakrishnan

Dr. Parikh Bhavna

Dr. Smruti B. K.

Oncosurgery

Dr. Bushan Kirti

Dr. Chabra Deepak

Di. Chabia Deepai

Dr. Jagannath P.

Dr. Katna Rakesh Dr. Parikh Deepak

Dr. Parikn Dec

Dr. Rao Satish

Dr. Sharma Sanjay

Dr. Shah Rajiv C. **Ophthalmology**

Dr. Agrawal Vinay

Dr. D'souza Ryan

Dr. Mehta Salil

Dr. Mehta Himanshu

Dr. Nagvekar Sandeep S.

Dr. Parikh Rajul

Dr. Shah Manish

Dr. Shah Sneha

Dr. Vaidya Ashish R.

Orthopaedic Surgery

Dr. Agrawal Pranav

Dr. Agrawal Vinod

Dr. Amyn Rajani

Dr. Archik Shreedhar

Dr. D'silva Domnic F.

Dr. Garude Sanjay

Dr. Joshi Anant

Dr. Kasodekar Vaibhav

Dr. Kodkani Pranjal

Dr. Kohli Amit

Dr. Mukherjee Sunirmal

Dr. Nadkarni Dilip

Dr. Nazareth Ritesh

Dr. Padgaonkar Milind

Dr. Panchal Lalit

Dr. Panjwani Jawahar S.

Dr. Shetty Nagraj

Dr. Vatchha Sharookh P.

Dr. Vengsarkar Nirad

Dr. Warrier Sudhir

Pathology

Dr. Chavan Nitin

Dr. Kamble Rahul

Dr. Mehta Kashvi

Dr. Natarajan Shripriya

Dr. Rangwalla Fatema

Paediatric Surgery

Dr. Bangar Anant

Dr. Karmarkar Santosh J.

Dr. Nathani Rajesh

Dr. Redkar Rajeev G.

Paediatrics

Dr. Chittal Ravindra

Dr. Gupta Priyam

Dr. Haria Kamlesh

Dr. Lokeshwar M. R.

Dr. Sharma Shobha Dr. Ugra Deepak

Paediatric Cardiology

Dr. Bhalgat Parag

Paediatric Critical Care/NICU

Dr. Arya Manish Kumar

Dr. Sheikh Minhaj Ahmed

Paediatric Endocrinology

Dr. Parikh Ruchi

Paediatric Hemato-Oncology

Dr. Kanakia Swati

Paediatric Neurology
Dr. Kulkarni Shilpa

Dr. Shah Krishnakumar N.

Paediatrics Nephrology

Dr. Ali Uma

Paediatric Opthalmology

Dr. Doshi Ashish

Paediatric Orthopedics

Dr. Aroojis Alaric

Paediatric Pulmonology

Dr. Khosla Indu

Pain Medicine

Dr. Baheti Dwarkadas

Dr. Jain Jitendra

Physicians / Internal Medicine

Dr. Ballani A. G.

Dr. Bandukwala S. M.

Dr. Dalvi Sunil G.

Dr. Gidwani Vinod N. Dr. Jadwani J. P.

Dr. Jadwani J. P. Dr. Medhekar Tushar P. Dr. Medhekar Amey T.

Dr. Nair C. C.

Dr. Shimpi Shrikant

Plastic & Reconstructive Surgery

Dr. Barve Devayani

Dr. Dixit Varun

Dr. Jain Leena

Dr. Kumta Samir

Dr. Nehete Sushil

Dr. Prakash Siddharth

Dr. Purohit Shrirang

Dr. Wagh Milind

Psychiatry

Dr. Deshmukh D. K.

Dr. Shah Bharat R.

Dr. Vahia Vihang N.

Psychology

Ms. Chulani Varkha

Physician / Rheumatology

Dr. Sangha Milan

Physiotherapy

Ms. Garude Heena

Radiology & Imaging
Dr. Deshmukh Manoj

Dr. Dhedia Khyati

Dr. Doshi Pankaj

Dr. Gupta Kanchan

Dr. Kamath Satish

Dr. Lokhande Kaustubh Dr. Mehta Mona

Dr. Tyagi Neha

Rehab Medicine

Ms. Shah Labdhi

Rheumatology

Dr. Chitnis Neena

Dr. Gill Niharika

Sleep Study Specialist

Dr. Samtani Anil

Spine Surgery

Dr. Bhojraj Shekhar

Dr. Chaddha Ram Dr. Kundnani Vishal

Dr. Mohite Sheetal

Dr. Nagad Premik

Dr. Nene Abhay

Dr. Patel Priyank

Dr. Varma Raghuprasad **Urology**

Dr. Pathak Hemant R.

Dr. Raina Shailesh

Dr. Raja Dilip

Dr. Sanghvi Nayan Dr. Shah Sharad R.

Dr. Vaze Ajit M. **Urological Laparoscopy Surgery**

Dr. Ramani Anup

Urodynamics Consultant

Dr. Dastur B. K. **Vascular Surgery**

Dr. Patel Pankaj Dr. Pai Paresh

TAVI (Transcatheter Aortic Valve Implantation)

What is TAVI?

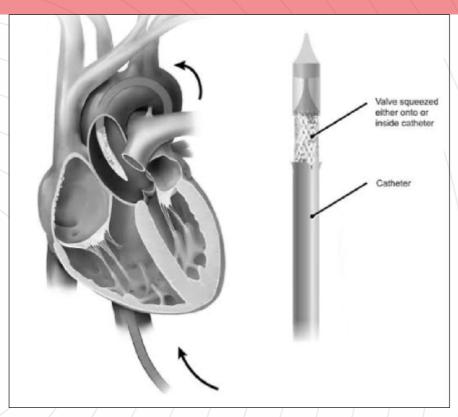
- It's a procedure that allows Aortic valve to be implanted using a catheter without the need for open heart surgery.
- It is considered as an innovative modality of treatment for inoperable cases and also now for low risk cases of Aortic Stenosis.

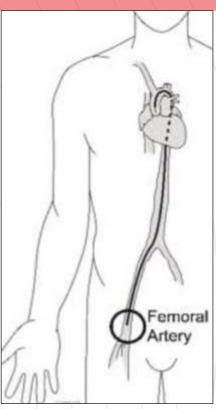
Benefits

- It is minimally invasive procedure
- Safe with excellent outcomes
- Hospital stay is considerably reduced to 4-5 days
- Faster recovery and return to normalcy

Why choose Lilavati Hospital?

- One of the few centres in Mumbai performing successful & safe TAVI procedures
- Highly experienced team of Cardiac experts
- State of the art technology and modern infrastructure
- Care at par with international standards





For queries call us on: 8291280428 (9 am - 5 pm)



Lilavati Hospital and Research Centre

More than Healthcare, Human Care

NABH Accredited Healthcare Provider

A-791, Bandra Reclamation, Bandra (W), Mumbai - 400 050. **Tel.:** +9122-2656 8000, +9122-2675 1000

Email: info@lilavatihospital.com • Website: www.lilavatihospital.com