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IN MANAGEMENT OF
HEART FAILURE

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MESSAGE FROM THE CHAIRMAN



Greetings!

It gives me great pleasure to present the latest edition of our hospital newsletter, a reflection of our continuous pursuit of excellence in healthcare, education, and service to the community.

At Dr. KM Cherian Institute of Medical Sciences, we remain steadfast in our commitment to providing compassionate, patient-centered care supported by clinical expertise and innovation.

I am particularly delighted that, beginning with this edition, our newsletter will feature department-focused articles, highlighting the remarkable work being carried out across various specialties.

This initiative aims to showcase the expertise, advancements, and patient care contributions of our departments while fostering greater knowledge sharing within our institution and community.

This edition shines a spotlight on our Cardiology and Cardiothoracic Vascular Surgery Departments, whose unwavering commitment to excellence continues to transform lives through advanced diagnostics, interventional procedures, complex cardiac surgeries, and comprehensive cardiac care. Their achievements stand as a testament to our mission of delivering world-class healthcare closer to home.

I extend my sincere gratitude to our patients and the community for their trust and confidence in us. Together, we will continue to build a healthier future.

Regards,

P. M. Sebastian

Chairman

Dr. KM Cherian Institute of Medical Sciences

MESSAGE FROM THE MANAGING DIRECTOR



Greetings!

Healthcare is a journey of continuous learning, innovation, and compassionate service.

At Dr. KM Cherian Institute of Medical Sciences, we strive each day to deliver exceptional care while advancing clinical excellence and fostering a culture of professional growth.

I am especially pleased to introduce our department-wise newsletter initiative, through which each edition will focus on a specific specialty area, showcasing its contributions, achievements, innovations, and impact on patient care.

In this edition, we proudly feature our Cardiology and Cardiothoracic Vascular Surgery Departments. These departments have consistently demonstrated excellence in managing complex cardiac conditions through advanced diagnostic services, interventional cardiology, cardiac surgery, rehabilitation, and multidisciplinary patient care. I extend my heartfelt appreciation to our doctors, nurses, allied health professionals, administrative staff, and support teams whose dedication makes these accomplishments possible.

As we look ahead, let us remain united in our vision of delivering compassionate, innovative, and high-quality healthcare while advancing our institution's legacy of excellence.

Together, let us continue building healthier lives and stronger communities.

Regards,

Fr. Dr. Alexander Koodarathil

Managing Director

Dr. KM Cherian Institute of Medical Sciences

COMPLEMENTARY AND ALTERNATIVE MEDICINES(CAM) IN MANAGEMENT OF HEART FAILURE



Dr. Anand Srinivasan
MBBS, MD, DM
Consultant Cardiologist

Complementary and alternative therapies has been traditionally used to define medical practices, products or systems that do not conform to standard beliefs of conventional medicine.

Complementary therapy-refers to non-mainstream approach used in combination with conventional medicine

Alternative therapy-Replaces conventional therapy altogether

Potential issues

1. Readily accessible
2. Very popular among patients because of misconceptions regarding their efficacy
3. Adverse effects are underemphasized
4. Can cause adverse interactions with conventional medications

This article is from AHA/ACC scientific statement with respect to the current evidence available regarding use of CAM in heart failure patients.

CAM therapies-4 domains

- 1-Biological based products-e.g. vitamins, fatty acids, prebiotics, probiotics etc.
- 2-Energy therapies-Reiki, sound healing
- 3-Manipulative and body-based methods-e.g. acupuncture, massage therapy
- 4 -Mind -body medicine-Relaxations, hypnosis, Yoga, Tai Chi etc.

Let's review the evidence for some of the common therapies used

Alcohol

Potential benefits include its positive effects of lipids, cardioprotective benefits of polyphenols of wines etc.- Some studies have shown benefits but no large-scale trials are available to make a definite recommendation in CHD

Co Q 10

In CHF there is ATP depletion. It is an antioxidant, cellular membrane stabilizer and Cofactor in many metabolic pathways, including oxidative phosphorylation. Exogenous supplementation of Co Q 10 has shown modest benefit in CHF patients. Trials have shown improvement in functional class and reduction in major adverse cardiovascular events at 2 years and possibly a reduction in all-cause mortality-Since large scale randomized trials are not available its use now is a Class 2 b ACC recommendation. Dose used is varied in different trials -A consensus is 200 to 300 mg per day in divided doses.

CAUTION-Co Q can decrease anticoagulant effect of Warfarin and can have additional blood pressure lowering effect when used with vasodilators.

Omega 3 fatty acids

REDUCE IT trial-showed significant cardiac benefits Used higher dose of up to 4 g of purified EPA (Eicosatetraenoic acid) However STRENGTH and



OMEMI trials did not show benefits These used EPA+DHA(Docosahexaenoic acid)
Difference in results are attributed to the different trial designs and the type of omega 3 used
CAUTION - A higher incidence of AF though a corresponding increase of strokes was not observed
Presently it is Class 2 b ACC recommendation

Yoga and Tai Chi

Can be used as an adjunctive wellness approaches to conventional medical

Vitamins

Vitamins-B1, C, D- have been studied. Routine supplementation is not beneficial but in patients with documented deficiency it may have some benefits

L carnitine

Trials show conflicting data, so it's not recommended by ACC as of now

In conclusion

None of the complimentary medicines have strong basis for recommendation in CHF so are not strongly recommended in treatment of CHF

However, treating physicians should be aware of the data available to counsel the patient and to look for any adverse effects to conventional therapy

Potentially beneficial medications among the available ones are CoQ10, L carnitine, Omega 3 FA, and Vitamins B1, D, C in deficient patients.



SUDDEN CARDIAC DEATH: SELECTED CLINICAL SUBSETS - INHERITED ARRHYTHMIC SYNDROMES



Dr. Devarajan K. A.
MBBS, MD, DM
Consultant Cardiologist

Sudden cardiac death (SCD) is defined as death due to cardiac cause that occurs within 1hr of onset of cardiac symptoms in witnessed cases or 24 hrs of last being seen apparently healthy and alive in unwitnessed cases. The incidence of SCD in general population is about 10.7 per 100,000 person years. Men are at a higher risk of SCD compared to age matched women (6.68 per 100,000 person years in men Vs 1.4 per 100,000 population in women). SCD in young adults (12-39 yrs) is 1.3 – 2.6 per 100,000 person years. Among athletes it is 0.24 – 6.8. Coronary Artery Disease is the underlying cause in 40% cases. The etiology of SCD can be broadly classified into (1) Structural heart disease (coronary artery disease, hypertrophic cardiomyopathy, Valvular Heart Disease, Dilated cardiomyopathy, Peripartum cardiomyopathy myocarditis, Arrhythmogenic cardiomyopathy etc). (2) Nonstructural (electrical). Long QT syndrome (LQTS) Short QT syndrome (SQTS), Catecholaminergic polymorphic tachycardia (CPVT) WPW syndrome etc). (3) Congenital and other causes (coronary artery anomalies, Aortic dissection, Commotio cordis etc). Emerging new or escalating factors contributing to SCD etiologies are (1) Post Covid sequele (myocardial inflammation and micro vascular thrombosis), (2) Substance related (opioids and stimulants) and metabolic syndrome in young adults. Inherited arrhythmogenic syndromes represent a significant cause of sudden cardiac death in young apparently healthy individuals. These diseases are characterized by high mortality risk often triggered by exercise. Often the first presenting symptom can be sudden cardiac death. Recent advances in genetic

testing and molecular imaging are re-shaping risk stratification. The emphasis is the need for comprehensive family screening and personalized management strategies to prevent SCD.

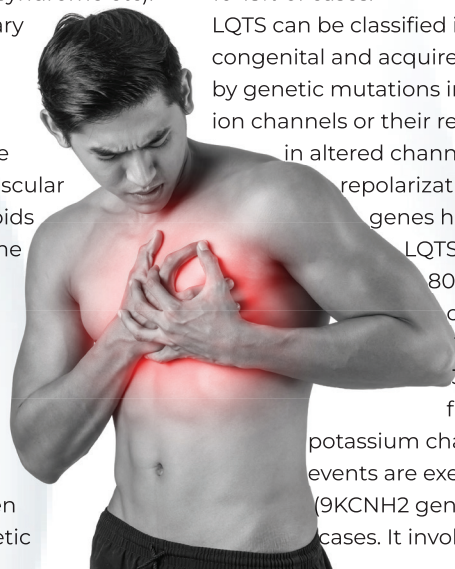
Long QT syndrome

Long QT syndrome (LQTS) is a rare, genetically heterogenous cardiac channelopathy characterized by prolonged ventricular repolarization, which manifest as a prolonged QT interval on surface ECG and increased risk of ventricular arrhythmias and SCD.

The prevalence of LQTS is estimated to be 1:2000 to 1:2500 in general population. The incidence of SCD varies depending on whether the patient receives treatment, their history of symptoms and their genetic type. Among untreated patients with h/o syncope, the mortality rate can be up to 20% per year, while in patients who receive appropriate therapy can be low (<1%). SCD can be first clinical manifestation in 10-15% of cases.

LQTS can be classified into two main categories – congenital and acquired. Congenital LQTS is caused by genetic mutations in genes encoding cardiac ion channels or their regulatory proteins resulting in altered channel function and prolonged repolarization. Mutations in at least 17 genes have been associated with LQTS.

LQTS types 1, 2 and 3 account for 80-90% of genotype positive cases. (1) LQT1 (KCNQ1 gene). The most common type, (30-35% cases) caused by loss-of-function mutation in the Iks potassium channel. Triggers for adverse events are exercise or swimming. (2) LQT2 (KCNH2 gene). Accounting for 25-30% of cases. It involves loss-of-function mutation



in the I_{Ks} Potassium channel. Events are often triggered by sudden auditory stimuli of emotional stress. (3) LQT3 (SCN5A gene), caused by gain of function mutation in SCN5A Sodium channel (5-10% of cases) characterized by events during sleep or rest and is considered the most lethal sub type.

Acquired LQTS results from various factors like (1) Medications like antiarrhythmic drugs, (class – I Quinidine, Procainamide and disopyramide, class III – e.g. Sotalol, dofetilide and ibutilide), Antibiotics like Erythromycin, Clarithromycin, Azithromycin, Fluoroquinolones like Levofloxacin, Antipsychotic drugs like Haloperidol, Thioridazine, Risperidone, Quetiapine and Ziprasidone, Anti-depressants like TCA (Amitriptyline, Imipramine, SSRI (Citalopram and Fluoxetine), Antimalarial like chloroquine Anti fungal (Ketoconazole, Fluconazole) Antiemetics (Ondansetron and granisetron), (2) Electrolyte imbalance (Hypokalemia, Hypomagnesemia, Hypocalcemia). (3) Cardiac conditions like bradycardia, myocardial ischemia and infarction, heart failure. (4) Endocrine disorders (hypothyroidism, Pheochromocytoma, (5) Neurological conditions like stroke, subarachnoid hemorrhage. (6) Liver diseases (Hepatic dysfunction), (7) Chronic Kidney disease, (8) Auto immune disorders (SLE).

The clinical presentation varies widely, ranging from completely asymptomatic individuals to those suffering sudden cardiac death as the initial symptom. Syncope occurs in approximately 60% of untreated patients, often caused by self-terminating episodes of Torsade-de-pointes (TDP). SCD can be the first presenting symptom up to 10% of untreated patients, especially in young otherwise healthy individuals. Seizures and palpitations resulting from transient ventricular arrhythmias are also common in patients with LQTS.

The incidence of SCD varies, in untreated symptomatic patients, who have history of syncope, SCD can occur in approximately 20% of patients with appropriate therapy (betablockers etc), annual mortality drops to less than 1% per year. Risk of SCD is more for type III LQTS, (20% Vs 4% for types I & II) and those with more prolonged QTc duration (>500ms). In 10-15% of patients, the first clinical manifestation can be SCD.

The mechanism of arrhythmogenesis in LQTS can be viewed as a two-stage process – Triggering and maintenance. (1) Early after depolarizations (EADs). The primary trigger for arrhythmia in LQTS is 'triggered activity' caused by EADs. Prolonged repolarization (long QT) keeps the cell membrane in a depolarized state for a longer period (Phase 2 or 3 of AP) and this provides enough time for L-type calcium channels to recover from inactivation and re-open. A sudden inward rush of calcium creates

a hump or secondary depolarization before the cell has fully reset, if this EAD reaches a certain threshold, it triggers a PVC – In LQT-1 EAD are typically triggered by sympathetic surges associated with physical activity. In LQT2, EAD are often triggered by sudden auditory stimuli or emotional stress. In LQT3 EAD occur most frequently at rest or during sleep (bradycardia dependent). (2). Transmural dispersion of repolarization (TDR). The heart is made of different cell layers. (Epicardium, endocardium, and M-cells) in LQTS M-cells experience much more prolonged AP prolongation than other layers. This creates a massive electrical mismatch across the heart wall. When an EAD triggered PVC occurs it may encounter tissue that is still refractory leading to unidirectional conduction block which allows the electrical signal to wander and spin, leading to the shift of QRS axes seen in TDP. (3) R from T Phenomenon – recent evidence from research show that the PVC is often physiologically born from the T-wave itself. The extreme delay in repolarization is so unstable that the T-wave literally sprouts the arrhythmia trigger.

Torsades-de-pointes (Td-P) is the hall mark arrhythmia in LQTS. It usually starts after a PVC and a short-long-short sequence. It can degenerate into VF leading to sudden cardiac arrest. Sustained monomorphic VT, Ventricular fibrillation, atrial fibrillation, and heart blocks also can occur in LQTS.

The Red flag signs of LQTS included: recurrent syncope or pre-syncope, especially on betablockers, exercise induced syncope triggered by emotional stress or loud noises etc., previous aborted SCD, family history of unexplained SCD in young individuals (<40 yrs. ECGs with extreme QTc prolongation (>500ms), T wave alternans, notched T waves in precordial leads 2:1 AV block and extreme bradycardia are considered high risk features.

Diagnosis of LQTS relies on evaluation of clinical history, family history, ECG findings and genetic testing. The Schwartz score is widely used to assess the probability of LQTS.

Non pharmaceutical management of LQTS involve avoidance of medications that can prolong the QT interval, avoidance of strenuous exercise and swimming and sudden noises. Prompt management of acute febrile episodes maintain good hydration and maintenance of optimum serum levels of Potassium and Magnesium. Surgical left cardiac sympathetic denervation can reduce the frequency of cardiac events by about 90%. Pharmaceutical management is primarily focused on reducing the risk of life-threatening arrhythmias such as TdP. Betablockers (Nadolol, propranolol) are the corner stone of treatment. They blunt the sympathetic stimulation, a common trigger for cardiac events. Betablockers are less effective for LQT-3. For patients with LQT3, class

Ib anti arrhythmia drugs, mexiletine has been shown to significantly shorten the QT interval and reduce arrhythmia events.

ICD implantation is considered for secondary prevention for survivors of aborted cardiac arrest (mandatory) and maybe recommended for those with recurrent syncope or Torsade-de-pointes despite maximal medical therapy (beta blockers + mexiletine) and cardiac sympathetic denervation. It carries a 40-60% complications for long term use (inappropriate shocks, lead fracture or infections).

Short QT syndrome

Short QT syndrome (SQTS) is a rare and life threatening inheritable channelopathy characterized by abbreviated corrected QT (QTC) interval on ECG and a significant risk of atrial and ventricular arrhythmia, potentially leading to SCD, in the absence of structural heart disease.

The true incidence and prevalence of SQTS is unknown. It is estimated to be less than one in 10,000 population. It can present at any age. There are 2 high risk peaks – In the first year of life and between 20 and 40 years. Males are affected more than females (75-84%).

The SQTS is primarily classified into different types based on the specific gene mutation and the ion channel affected. The types are generally divided into 2 categories. Mutations that cause gain-of-function in Potassium channels (Types 1-3) and mutations that cause loss-of-function in Calcium or Sodium channels (Types 4-8). SQTS is almost always inherited in an autosomal dominant manner. Only about 20-25% of people diagnosed will have a positive genetic test. Common genetic mutations include KCNH2 (SQT-1), KCNQ1 (SQT-2), KCNJ2 (SQT3), CACNA1C, SCN 5A (rare) etc.

Clinical presentation of SQTS is variable, ranging from asymptomatic individuals to those with palpitations, syncope or life-threatening arrhythmias such as atrial fibrillation (70-80%) polymorphic VT or ventricular fibrillation and SCD. Events often occur at rest or sleep rather than during physical activity.

The mechanism of arrhythmogenesis in SQTS involves significant shortening of the cardiac action potential duration and the effective refractory period (ERP).

The shortened ERP reduces the cardiac excitation wavelength which allows for small re-entrant circuits facilitating the maintenance of circus type or spiral wave re-entry leading to a trial or ventricular fibrillation. Non uniform shortening of the action potential across different layers of myocardium results in increased transmural dispersion of repolarization where premature beats can encounter regions of functional block initiating re-entry.

The hall mark of SQTS is a shortened corrected QT

interval (QTC). A definite diagnosis can be considered when the QTC interval is <340ms on a resting ECG with heart rate <80bpm. Diagnosis may also be considered at a QTC <360ms, if accompanied by specific clinical features like (1) A confirmed pathogenic mutation in a known SQTS associated gene (like KCN H2, KCNQ1, KCNJ2), (2) A family history of confirmed SQTS or SCD at age <40 yrs, (3). Survival from a documented VT / VF episode without structural heart disease. The dollop scoring system is used to assess the probability of SQTS based on ECG features, clinical history, family history and genetic features. Diagnosis requires the exclusion of secondary causes of SQT like electrolyte imbalance (Hypercalcemia, Hyperkalemia), Hypothermia, drug induced effects or genetic conditions like primary carnitine deficiency.

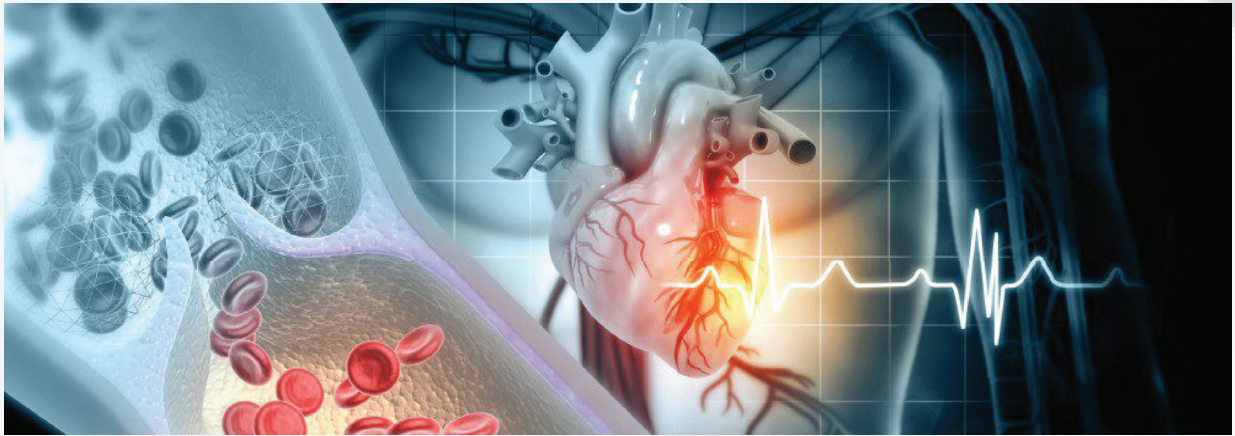
The red flag signs of SQTS are (1) sudden cardiac arrest – the most alarming sign of SQTS. (2) Syncope – can occur in 25% of patients. It is a sign of underlying malignant arrhythmias. (3) Early onset AF, in children or young adults. (4) Frequent palpitations reported in 30% of patients. (5) SQT may be a cause for sudden infant death syndrome (SIDS) under 1 year old. High risk features in ECG are (1) abnormally short (<340ms) QTC interval, (2) Absent or short ST segment. (3) Tent in the desert T waves as in hyperkalemia, and PQ segment depression.

Pharmacological management of SQTS primarily aims to prolong QT interval and prevent life threatening arrhythmias. Quinidine / Hydroquinidine is considered the most effective pharmacological agents (Especially for SQTS-1) Disopyramide also can prolong QT interval and the ventricular effective refractory period.

The corner stone of management of SQTS is implantation of ICD. For secondary prevention, ICD is recommended for patients who have survived a cardiac arrest or have documented sustained VT or VF. For primary prevention, ICD may be considered for patients with SQTS, with a family history of SCD at young age or those who have unexplained syncope, that is suspected to be arrhythmogenic. Concerns about long term ICD use are inappropriate shocks due to over sensing of tall T waves, risks of lead fracture, displacement or infection and psychological impacts related to these factors.

Brugada Syndrome

Brugada syndrome (BrS) is a rare disorder characterized by coved or saddle shaped ST segment elevation in the right precordial leads on ECG. It is associated with increased risk of ventricular arrhythmias such as ventricular tachycardia / Ventricular fibrillation (VT / VF) which can lead to SCD.



Brs has three ECG patterns. Type I shows a coved ST segment elevation while Type II and III are saddle back shaped. Type II having at leads 2mm ST elevation and Type III having less than 1mm. The expression of Brugada pattern on the ECG may occur only when triggered by factors like, fever, medications, or alcohol use etc. Ajmidine, a Sodium channel blocker given intravenously can be used to unmask the hidden ECG abnormality of type I Brs, in suspected cases.

Overall global prevalence of Brs is 5/10,000 in general population. Incidence in men is 8-10 times that of women. Clinical manifestations usually appear in adults (3rd and 4th decades) lifetime risk of syncope / SCD as a result of VTA has been reported to be between 10-15%. Approximately 38% of patients presenting with SCD have a previous h/o syncope. Initially Brs has been perceived as an electrophysiological disorder, but recently structural abnormalities such as myocardial atrophy, fibrosis and lipid infiltration was found in RVOT, which may contribute to arrhythmogenesis.

Brugada is an inherited channelopathy. It is predominantly inherited in an autosomal dominant manner. It is caused by mutations in the genes that control Sodium (20-30% cases), Potassium and Calcium channels (5% cases), that regulate the electrical rhythm. There is 50% chance to develop Brs if one of the parents has the mutation. For 65-70% of patients with Brs, no specific gene mutation can be detected. Both men and women have equal chance of inheriting the gene but the disease is 8-10 times more common in men due to hormonal influences. There is variable expressivity within the same family with mutant genes.

The primary clinical presentation of Brs include sudden cardiac death, which can be the first presenting symptom especially in young adults (can occur during sleep or rest). Syncope occurs in approximately 1/3 of diagnosed patients and is considered a significant risk factor for future cardiac events if it is due to arrhythmias. Nocturnal agonal respiration (gasping and labored breathing) can occur

due to self-terminating VF. Palpitations, dizziness, or seizures often occur due to underlying cardiac arrhythmias.

Recent researches suggest two important mechanism of arrhythmogenesis. (1) The repolarization hypothesis. It consists of a loss-of-function mutation in the SCN5A gene which reduces the inward Sodium current (I_{Na}). This allows the transient outward Potassium current (I_{to}) to dominate especially in the RV epicardium. This imbalance leads to a notched or completely lost action potential (AP) dome in the epicardium, while the endocardial AP remains relatively normal. The resulting voltage gradient between epicardium and endocardium causes ST segment elevation and allows phase-2 re-entry. When the AP propagates from the endocardium back into the repolarized epicardium, triggering of PVCs can occur. (2) In the depolarization hypothesis, arrhythmias are attributed to conduction slowing rather than repolarization differences. (often localized to RVOT epicardium). Delayed activation in the RVOT relative to the rest of the ventricle create a slow conduction zone. This delay provide a substrate for circus movement, re-entry resulting in arrhythmias. (3) The neural crest hypothesis. It suggests that Brs is an 'arrhythmogenic cardiomyopathy' restricted to the RVOT. The syndrome arises because of the neural crest derived tissue in the RVOT, which remains structurally and electrically distinct from the rest of the heart creating an environment for re-entry.

Common arrhythmias in Brs are (1) VF. The most dangerous arrhythmia which may result in SCD. It can occur during rest or sleep. (2) Polymorphic VT. Usually initiated by short coupled PVCs often self-terminated but can degenerate into VF. (3) Atrial fibrillation. It is the most common arrhythmia associated with Brs. (10-25% of patients). (4) Monomorphic VT. Rare often associated with SCN5A mutation. (5) Brady arrhythmias (AV Blocks and sick sinus syndrome) and conduction defects are also reported associated with Brs.

Common triggers of Brs include fever, medications (class I anti arrhythmic drugs, Tricyclic Antidepressants, certain anesthetics) increased vagal tone (occur at rest or during sleep), heavy meals, alcohol consumption, electrolyte disturbances (hyper and hypokalemia) etc.

Diagnosis of Brs is based on the presence of Type – I pattern on ECG. In the absence of spontaneous type – I pattern (type II and III) Pharmacological challenge tests using Sodium channel inhibitors (such as ajmalin), Procainamide or Flecainide.

The Red flag signs in Brs are (1) Arrhythmic syncope is the most significant clinical red flag. Syncope can occur at rest, during sleep or while having a febrile illness. (2) Nocturnal Agonal Respiration (gasping or labored breathing) often due to a self-terminating arrhythmia. (3) Aborted cardiac arrest. It is the strongest predictor of future lethal events. (4) Family history of SCD in a first degree relative under the age of 45 yrs. (5) ECG features like spontaneous type I (coved) pattern fragmented QRS, aVR sign (prominent, 3mm R Waves suggesting conduction delay in RVOT) early repolarization etc.

Lifestyle modification are important in the management of Brs. Febrile episodes should be managed aggressively. Alcohol and drugs that can trigger VTA to be avoided. High intensity competitive sports to be avoided. Large carbohydrate – heavy meals especially before sleep also can trigger arrhythmias.

Quinidine is the most extensively studied pharmacological agent for management of Brs. It blocks the transient outward Potassium current (Ito) and help to rebalance the action potential. It is used to prevent VTA in patients with recurrent shocks or who cannot receive or refuse an ICD. Other medications which can be tried are Cilostazole, Bepridil and Disopyramide (limited data).

In the event of an electrical storm, intravenous isoprenaline is recommended. Certain drugs can unmask the Brugada pattern and can trigger VTA. Anti-arrhythmic drugs like, Flecainide /Ajmaline and procainamide, Anaesthetic agents like propofol, Bupivacaine, Psychotropic drugs like Amitriptyline, Lithium and clomipramine to be avoided.

Patients who have survived SCD due to VF or pulseless VT or those who have documented sustained VT / VF with symptoms like syncope are recommended for ICD implantation (Class I) for primary prevention, ICD may be considered for patients with Type I ECG pattern plus risk factors like unexplained syncope (presumed to be arrhythmogenic) family h/o premature SCD, induction of VT / VF during electrophysiology study (Class II).

Catecholaminergic Polymorphic Ventricular

Tachycardia

Catecholaminergic polymorphic Ventricular tachycardia (CPVT) is a rare genetic cardiac channelopathy that can cause life threatening ventricular tachy arrhythmias (VT / VF) especially during physical activity or emotional stress. Patients generally have structurally normal heart and normal resting ECG.

The incidence of CPVT is estimated to be affecting in one in 10,000 individuals. It is responsible for up to 15% of unexplained SCD in young people (1-35yrs).

Mean age of onset of symptoms range from 7-12 yrs (60% of people experience their first symptoms or cardiac arrest by the age of 20 yrs), 30% of patients have positive family history of stress related syncope / seizure / SCD in relatives under the age of 40 yrs. CPVT affects males and females equally.

CPVT is mostly inherited as an autosomal dominant fashion. Most cases of mutations occur in the gene responsible for encoding the cardiac ryanodine receptor (RYR2) in 60% of cases or in the calsequestrin – 2 genes (CASQ2) disrupting the calcium ion handling within the cardiac myocyte sarcoplasmic reticulum. The mutation in RYR2 results in abnormal calcium handling characterized by excessive accumulation of diastolic calcium in the sarcoplasmic reticulum and spontaneous release into the cytoplasm. This leads to delayed after depolarizations and triggered activity, ultimately resulting in malignant ventricular arrhythmias especially under conditions of physical stress or emotional excitement since catecholamine mediated beta-adrenergic receptor activation can trigger premature sarcoplasmic reticulum calcium release.

Symptoms usually begin in childhood or adolescence and include palpitations, dizziness, syncope, or cardiac arrest due to VT or VF often triggered by emotional or physical stress. Two different types of polymorphic VT can occur. (1) Typical polymorphic VT and (2) Bidirectional VT (rare). About 40% of patients die within 10 yrs, if untreated.

A definite diagnosis is typically established based on adrenergic – induced ventricular arrhythmias (Polymorphic / bidirectional VT) in patients with structurally normal heart with normal resting ECG. In clinically suspected patients, exercise stress test is the primary diagnostic method to confirm diagnosis. Exercise can provoke NSVT or sustained VT (in 63% of patients with CPVT). When routine stress test fails to demonstrate VTA, a burst exercise protocol may be used. For those patients who are unable to do exercise stress test epinephrine infusion can be tried. In younger patients, Holter monitoring may be tried (has only less sensitivity).

Genetic testing is recommended for all individuals with clinically suspected CPVT. Knowing the specific

gene involved and the location of the variant may help in risk assessment and its management decisions.

For symptomatic patient's aggressive pharmacological management should be done. Lifestyle modification is important for all affected individuals. Strenuous physical activity and competitive sports should be avoided. Stress inducing environment also should be avoided.

Betablockers are the corner stone in the management of CPVT to prevent arrhythmic events. Nadolol and Propranolol (Nonselective betablockers) are preferred and to be administered to the maximum tolerated dose. For those patients who have symptoms in spite of maximum betablocker therapy, Flecainide effectively reduce symptoms when given as an add on therapy.

Non pharmacological therapies like left cardiac sympathetic denervation (LCSD) is an effective ant fibrillatory intervention, for whom pharmacological intervention are proved ineffective.

ACC/AHA/HRS guidelines and recent ESC guidelines provide a class I indication for ICD for patients with history of aborted cardiac arrest. ESC guidelines provide a class II A recommendation for ICD in patients who experience arrhythmogenic syncope and / or demonstrated bidirectional or polymorphic VT, while on highest tolerated dose of Betablockers and Flecainide. But usage of ICD for CPVT is controversial. Inappropriate ICD shocks can occur more frequently (20-30% of patients with CPVT). Stress due to these shocks itself can be a trigger for arrhythmic episodes. ICD shocks to convert polymorphic VT and bidirectional VT to sinus rhythm are generally ineffective. ICD related complications are high since this device is implanted in younger individuals (29%). It may not provide mortality benefit in all cases. So presently the expert opinion is to reserve ICD placement as a last resort when the combination of betablocker, flecainide and LCSD has proven ineffective. Recent studies have shown that catheter ablation of PVCS inducing VF hold promise for preventing these fatal arrhythmias.

Conclusion

SCD resulting from inherited arrhythmias such as LQTS, SQTS, Brugada syndrome, CPVT etc, represent a catastrophic event often occurring in young asymptomatic individuals, with non-significant structural abnormalities. These conditions represent a significant challenge in risk stratification. The evolution of molecular genetics now enables identification of high-risk individuals. The corner stone of prevention relies on comprehensive family screening, through 12L ECG

analysis and implementation of genetic testing following unexplained cardiac arrests (particularly in young patients). A multidisciplinary approach involving cardiologists, genetic counsellors and electrophysiologists is essential to interpret complex results and decide on protective strategies like betablocker therapy or ICD implantation.

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AT THE EDGE OF STABILITY: A CLINICAL NARRATIVE FROM CARDIAC SURGERY



Dr. Sunil Agarwal
MBBS, MS, MCh (CVTS),
DNB (CVTS)
Consultant Cardiologist



Dr. Mithun Varghese Mathew
MBBS
Junior resident CTVS



Dr. Sharefa Nazar
MBBS
Junior resident CTVS

Abstract

Native coronary artery spasm after coronary artery bypass grafting is a rare but life-threatening condition. The incidence of perioperative coronary artery spasm (CAS) after on-pump or off-pump coronary artery bypass grafting (CABG) is relatively rare, with reported rates ranging from 0.4% to 1.3%. Some broader studies suggest it may occur in up to 1% to 8% of patients. Herein, we report the case of a 64-year-old man who received on pump coronary artery bypass grafting. Soon after surgery before shifting to the ICU, there was hemodynamic compromise due to right coronary artery spasm, prompting emergent coronary angiography with Intra-aortic balloon support. Because the angiography results showed severe spasm of the entire native right coronary artery, the patient was treated with an intracoronary nitro-glycerine followed by stenting. The patient recovered in 14 days with mechanical support, catecholamines. Although coronary artery spasm after on-pump coronary artery bypass surgery is a rare condition, it must be suspected when sudden circulatory collapse occurs.

Case Presentation

A 67-year-old gentleman with a history of diabetes mellitus and systemic hypertension presented with

complaints of effort angina (CCS Class II). Evaluation revealed a positive treadmill test suggestive of inducible myocardial ischemia. Echocardiography demonstrated mild mitral regurgitation with good left ventricular systolic function. He had previously undergone PTCA to LAD one month prior to second presentation. Re CAG showed 70% late loss in LAD DES. His coronary angiogram revealed LAD with diagonal 70% involvement. A heart team discussion concluded that surgical revascularization would provide the most durable outcome, given his overall risk profile. He was planned for surgical revascularization after completion of routine preoperative investigations and obtaining all necessary clearances. After detailed counselling regarding the risks, benefits, and expected outcomes of surgery, informed consent was secured from the patient and relatives. He was then taken up for elective coronary artery bypass grafting under general anaesthesia. Following sterile preparation and median sternotomy, the chest was opened in the standard fashion and the LIMA was harvested meticulously. Sequential LIMA grafting to the LAD and diagonal artery was performed successfully with satisfactory distal flow across the grafts. The procedure progressed smoothly in an uneventful manner, with stable hemodynamic and cardiac function maintained throughout.



With all planned grafts completed and the heart recovering well, the surgery appeared to have gone well, and preparations were done for routine postoperative transfer -everything remained stable, until it was not. 'ST elevations in the inferior lead'.

RCA territory - a vessel previously reported as normal on both the initial CAG and repeat CAG, findings that had been independently reviewed by both cardiologists and surgeons. Was this merely postoperative pericarditis? Reciprocal ECG changes? A transient coronary steal phenomenon as the LAD was smaller than the diagonal causing flow compromise to LAD leading to ECG changes? Graft occlusion? In view of persistent inferior lead ST-segment elevations with worsening hemodynamic instability, the sternal wires were soon cut, and the chest reopened.

The patient was placed back on cardiopulmonary bypass and graft revision was performed in view of ongoing concern for graft occlusion. The LAD was regrafted using a saphenous vein graft, and the LIMA was grafted to the diagonal artery. Despite revision of the grafts, significant hemodynamic instability persisted, and the patient could not initially be weaned from cardiopulmonary bypass. An intra-aortic balloon pump (IABP) was therefore inserted for mechanical circulatory support, following which the patient was gradually weaned off bypass.

However, hours passed, and the ST-segment elevations continued to persist. Cardiologists were called into the operating room, and multiple discussions were held between the cardiology and surgical teams, as the cause of the ongoing ischemic changes remained unclear despite graft revision and mechanical support. In view of persistent instability and unresolved ECG changes, an emergency decision was made to shift the patient to the Cath lab for urgent check angiography under IABP support. The entire operative team accompanied the patient during transfer. Check angiography demonstrated a patent LAD graft with good distal flow. Although the RCA had been reported as normal on previous angiograms and was initially not suspected to be the culprit vessel, repeat angiography of the RCA was nevertheless performed.

As the dye flowed through the vessel, the diagnosis finally became evident - severe right coronary artery spasm.

During check angiography, the patient developed profound hypotension and bradycardia, requiring immediate resuscitative measures with emergency medications and continued hemodynamic support. In view of the persistent RCA spasm, the cardiology team initially advised surgical grafting to the RCA; however, the surgical team remained hesitant given the previously normal native vessel and the possibility of competitive flow compromising long-term graft patency. Intracoronary nitro-glycerine was administered in an attempt to relieve the spasm, but the spasm continued to persist. After discussion, a decision was made to proceed with percutaneous intervention to the RCA. A coronary stent was deployed successfully across the spastic segment with subsequent restoration of satisfactory flow and gradual resolution of the ischemic changes. In view of the prolonged instability and ongoing postoperative concerns, the chest was left open and the patient was shifted to the CSICU with IABP support. Over the following 48 hours, the ST-segment elevations settled, and the patient gradually achieved hemodynamic stability. The IABP was subsequently removed, following which chest closure was done successfully. The patient showed steady clinical improvement thereafter and was later shifted to the ward in stable condition. He was eventually discharged with advice for regular follow-up. During subsequent follow-up, he required readmission for complaints of generalized weakness, melena, and low haemoglobin levels, which were managed conservatively with supportive care and medical management.

Discussion

In the context of acute postoperative circulatory instability, prompt recognition and targeted intervention for coronary vasospasm are vital. In this case, emergency coronary angiography, prompted by hemodynamic instability and ST elevations in Inferior leads, unveiled diffuse coronary artery spasm, however the RCA spasm was managed by stenting in view

of severe spasm as there was always an uncertainty about it recurring later both in late post operative recovery period in the hospital or even after discharge from the hospital. To avoid this uncertainty, the team decided to proceed with stenting rather than grafting. The concern was that if grafting had been performed, once the spasm subsided, the graft might not have been able to compete with the restored native coronary flow, creating a risk of graft occlusion. Therefore, preserving the native coronary flow with stenting was considered the most appropriate option. Despite its rarity, if not swiftly addressed, coronary vasospasm following cardiac surgery can be fatal. The use of the intra-aortic balloon pump is crucial for enhancing coronary flow in severe cases, and the employment of ECMO provides critical support in instances of severe cardiogenic shock or cardiac arrest, effectively addressing left ventricular dysfunction and associated metabolic disturbances.

Conclusion

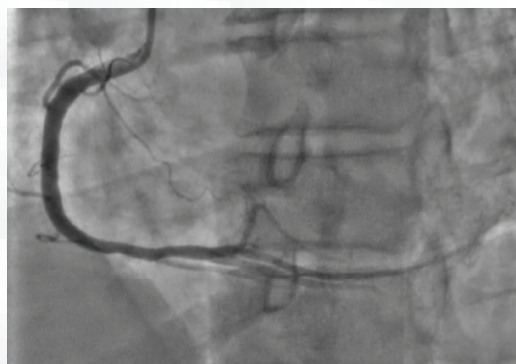
In conclusion, this case highlights the necessity of considering postoperative coronary spasm as a potential cause of severe cardiogenic shock or cardiac arrest. Additionally, it underscores the crucial role of coronary angiography in diagnosing and managing this condition. Such experiences reinforce that in cardiac surgery, outcomes are not defined solely by the procedure performed, but by the team's ability to respond effectively when the clinical course deviates from expectation.

CORONARY ANGIOGRAPHY REPORT	
• LMCA	: - Normal.
• LAD	: - Type III. After D1 discrete 90% stenosis.
• DIAGONALS	: - Normal.
• LCX	: - Non dominant. Normal.
• OMs	: - Normal.
• RCA	: - Dominant. Normal.
• PDA	: - Of RCA – Normal.
• PLB	: - Of RCA – Normal.
> IMPRESSION	: - SVD.
> ADVICE	: - PTCA to LAD.

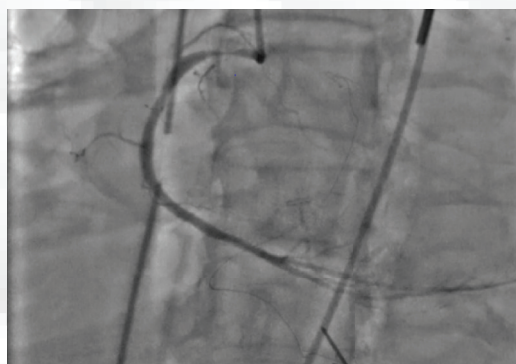
CORONARY ANGIOGRAPHY REPORT	
• LMCA	: - Normal.
• LAD	: - Type III stent in situ 70% - late loss in Mid LAD DES.
• DIAGONALS	: - D ₁ – Ostial 70% stenosis.
• LCX	: - Non dominant and normal.
• OMs	: - Normal.
• RCA	: - Dominant.
• PDA	: - Of RCA – Normal.
• PLB	: - Of RCA – Normal.
> IMPRESSION	: - SVD – 70% late loss in LAD DES.
> ADVICE	: - DEB to LAD DES / CABG with grafts to LAD & D ₁ .

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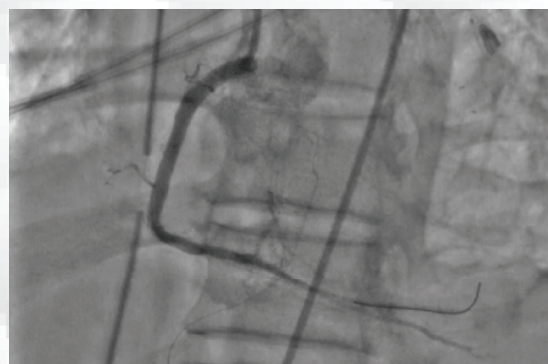
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Preoperative coronary angiogram images revealing normal Right coronary arteries.



Immediate Post op Check Angio revealing ostial RCA spasm



Post Stenting after RCA spasm

CORE STORY

THE BEAUTY OF AN ORCHID IS A SOURCE OF JOY AND LIVELIHOOD FOR MANY.



Dr. Rakhi K. R.
MBBS, MD, PDCC, FCCM
Consultant Cardiac Anesthetist & Intensivist

From the hum of the operating room to the quiet glow of a laminar flow hood I found two worlds that demanded the same things: steady hands, meticulous technique, and the courage to steward life through fragile beginnings. By day, I navigate the high-stakes rhythm of cardiac anesthesia—reading monitors, calibrating doses, and keeping hearts steady when lives hang in balance. By night and between cases, I return to another kind of life support: a tissue culture lab where tiny orchid protocorms unfurl their first leaves under sterile light.

My fascination began as a refuge from the pressure of medicine. What started as a hobby—rescuing a sick plant, coaxing a stubborn seedling—grew into a disciplined practice. I brought surgical rigor to plant science: aseptic technique learned in the OR translated into contamination-free cultures; patience sharpened by long surgeries translated into months of careful subculturing.

Over years, I built reproducible protocols for high frequency micropropagation, mastering flask culture, ex-agar acclimatization, and producing robust mature plants ready for potting.

But my dream always reached beyond lab success. I imagined an enterprise that combined scientific excellence with deep social impact: a vertically integrated orchid venture producing healthy mature plants, sealed flasks of cultured orchids, and ex-agar plantlets ready for sale. I saw a supply chain that started in sterile vessels and ended in living green businesses across Kerala and beyond.

Central to that vision was a commitment to community—especially to women. I plan to partner with Kudumbashree as a deliberate social design: recruit and train women as growers, caregivers, and sales agents. In structured training modules, I teach aseptic handling for ex-agar plants, routine nursery practices for potting and hardening, and customer-facing skills for market stalls and online sales.



Kudumbashree groups operate micro-nurseries under my quality protocols, receiving starter flasks and technical support while earning production-based pay and a share of retail margins. This model produces three products suited to different customers and scales:

1. Mature orchids for retail and events, grown in community nurseries and sold locally.
2. Flasks of sterile cultures for hobbyists and research buyers, prepared in my central lab.
3. Ex-agar plantlets, transitioned from lab to nursery and sold as affordable, low-maintenance plants.

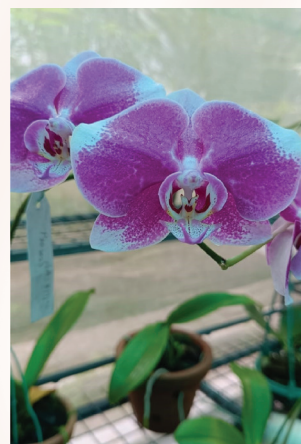
The enterprise scales locally while building export readiness. I implement traceability and phytosanitary

best practices from the outset—clean-room standards, standardized labels, batch records, and documentation needed for phytosanitary certification. Early partnerships with local exporters and guided trials prepare Kudumbashree growers for the higher standards of international buyers. Profits are reinvested to expand lab capacity, supply cold-chain logistics, and subsidize training for more women across districts.

Beyond income, the program transforms lives. Women who join gain technical skills, steady pay, and business literacy. Micro-nurseries become community hubs where knowledge spreads—students learning about plant science, neighbors buying plants, festivals showcasing local orchids. The enterprise becomes a local brand associated with quality orchids and women's empowerment.

My identity as a cardiac anesthesiologist remains vital to the story. Medical credibility opens doors—providing discipline in documentation, networks for funding, and an ethical framework for mentorship. It also shapes my leadership: I can run the business like a clinical trial—define endpoints, measure outcomes (survival rates of ex-agar plants, contamination rates, sales per grower), I create protocols, and scale what works.

Years ahead, the company will be known for export-grade orchids shipped from Kerala to specialty markets abroad, with a clear social footprint: thousands of women employed or upskilled, dozens of thriving micro-nurseries, and a replicable model adopted in neighboring districts. When asked about success, I point not only to revenue or scientific advances but to the sight that first inspired me—women in bright sarees tending trays of orchids, confident and skilled, transforming sterile flasks into living livelihoods.



Endovascular Management of Traumatic Thoracic Aortic Injury in a Young Male Following Road Traffic Trauma



Dr. Jaيمي Abraham

MBBS, MD (Radiodiagnosis), Fellowship in Vascular Interventional Radiology (FVIR), European Board of Interventional Radiology (EBIR)

Consultant Interventional Radiologist

Abstract

Background

Blunt thoracic aortic injury (BTAI) is one of the most lethal consequences of high-energy blunt trauma and remains a significant cause of trauma-related mortality. Advances in imaging and endovascular technology have transformed the management paradigm, with thoracic endovascular aortic repair (TEVAR) increasingly preferred over open surgical repair due to lower morbidity and mortality. We report a case of SVS Grade III BTAI associated with type B aortic dissection successfully managed using TEVAR.

Case Presentation

A young male presented to the emergency department following an autorickshaw rollover accident. Contrast-enhanced CT aortography revealed a Society for Vascular Surgery (SVS) Grade III blunt thoracic aortic injury characterized by a

pseudoaneurysm of the descending thoracic aorta with associated Stanford type B aortic dissection. Following multidisciplinary discussion involving Interventional Radiology, Cardiothoracic and Vascular surgery, Anaesthesia and Critical Care teams, the patient underwent urgent Thoracic Endovascular Aortic Repair (TEVAR). Endovascular exclusion of the injured aortic segment was successfully achieved with complete sealing of the lesion and preservation of distal perfusion. The postoperative course was uneventful. The patient was discharged in a stable state. Follow-up imaging after 1 month demonstrated satisfactory stent graft position without endoleak.

Conclusion

This case highlights the pivotal role of interventional radiology in contemporary management of aortic emergencies. TEVAR provides a minimally invasive, effective, and durable treatment option for traumatic thoracic aortic injuries while emphasizing the importance of multidisciplinary collaboration in achieving favourable patient outcomes.

Introduction

Blunt thoracic aortic injury (BTAI) represents the second most common cause of death following blunt trauma, exceeded only by traumatic brain injury. Most patients succumb before reaching medical care, making prompt diagnosis and treatment critical for survival. The aortic isthmus is the most affected site because of its relative fixation by the ligamentum arteriosum and susceptibility to deceleration forces. Historically, open surgical repair was considered the standard treatment for significant BTAI. However, over the past two decades, TEVAR has emerged as the preferred treatment modality due to lower perioperative mortality, reduced incidence of paraplegia, and shorter recovery times. The Society for Vascular Surgery (SVS) classifies BTAI into four grades, with Grade III injuries characterized by pseudoaneurysm formation generally warranting definitive repair.

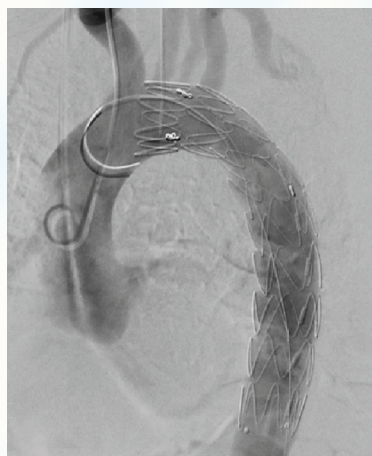
We report a young male patient with SVS Grade III BTAI and associated Stanford type B aortic dissection following an autorickshaw rollover accident,



Intimal flap?
Contained Pseudoaneurysm.



SVS Type 3 Aortic injury with Aortic Dissection



Post stenting Aortogram showing complete exclusion of the contained pseudoaneurysm and aortic dissection.

successfully managed with TEVAR through close collaboration between interventional radiology and cardiothoracic surgery teams.

Case Presentation

A young male presented to the emergency department after sustaining blunt thoracic trauma during an autorickshaw rollover accident. On arrival, the patient was conscious and hemodynamically stable.

Primary trauma survey was performed as per hospital guidelines. Given the mechanism of injury, a contrast-enhanced CT trauma evaluation including CT aortography was obtained.

CT angiography demonstrated:

- * Focal traumatic injury involving the proximal descending thoracic aorta at the aortic isthmus.
- * Pseudoaneurysm formation consistent with SVS Grade III blunt thoracic aortic injury.
- * Associated Stanford Type B aortic dissection.
- * No evidence of active extravasation.
- * No signs of rupture or mediastinal hematoma progression.

Following multidisciplinary consultation involving Interventional Radiology, Cardiothoracic and Vascular Surgery, Anaesthesia and Critical Care teams, endovascular repair was deemed the most appropriate treatment strategy.

Intervention

The procedure was performed in the Cath lab suite under fluoroscopic guidance.

Common femoral arterial access was obtained, and thoracic aortography from right brachial access confirmed the CT findings. Appropriate measurements were performed to determine the proximal and distal landing zones.

A thoracic endograft was deployed across the injured aortic segment, covering the left subclavian artery, site of pseudoaneurysm and entry tear. Completion angiography demonstrated:

- * Complete exclusion of the pseudoaneurysm.
- * Successful sealing of the dissection entry site.
- * No evidence of type I or type III endoleak.
- * Preserved flow through the distal thoracic and abdominal aorta.

- * Good cerebrovascular blood flow.

The procedure was completed without immediate complications.

Outcome and Follow-Up

The patient remained hemodynamically stable postoperatively and was monitored in the intensive care unit.

Patient is under constant follow-up for any left upper limb symptom development as the left subclavian artery was covered by the stent graft. The patient is planned for a left carotid – subclavian artery bypass after plugging the left subclavian artery via left brachial artery access, if any symptoms / endoleak occur later.

Follow-up CT angiography done after 1 month demonstrated:

- Stable stent graft position.
- Complete exclusion of the pseudoaneurysm.
- No endoleak.
- Satisfactory remodelling of the thoracic aorta.
- Good flow into the left subclavian artery.

Discussion

BTAI occurs following rapid deceleration injuries commonly encountered in road traffic accidents, falls from height, and crush injuries. The aortic isthmus is particularly vulnerable due to differential mobility between the aortic arch and descending thoracic aorta.

The Society for Vascular Surgery classification includes:

Grade	Description
I	Intimal tear
II	Intramural hematoma
III	Pseudoaneurysm
IV	Rupture

Current guidelines recommend repair of Grade III and Grade IV injuries, while selected Grade I and II injuries may be managed medically.

The introduction of TEVAR has revolutionized treatment of thoracic aortic injuries. Multiple studies have demonstrated:

- Lower mortality compared with open repair.
- Reduced paraplegia rates.

- Reduced stroke rates.
- Shorter intensive care and hospital stay.
- Faster recovery.

This case also underscores the growing role of interventional radiology in aortic emergencies. Modern IR services provide rapid image-guided diagnosis, procedural planning, endovascular expertise, and postoperative imaging surveillance. In many centres, interventional radiologists play a central role in the management of traumatic vascular injuries alongside vascular and cardiothoracic surgeons.

Equally important is the multidisciplinary approach. Successful treatment in this patient was achieved through coordinated decision-making between interventional radiology, cardiothoracic surgery, anaesthesia, emergency medicine, and critical care teams. Such collaboration facilitates optimal patient selection, procedural planning, perioperative management, and follow-up care.

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One-month follow-up CT Aortogram showed

- Stable stent graft position.
- Complete exclusion of the pseudoaneurysm.
- No endoleak.
- Satisfactory remodeling of the thoracic aorta.
- Good flow into the left subclavian artery.

ECHOCARDIOGRAPHY THE LUB-DUB



Mrs. Suja Kurian
In charge
Clinical Technicians

The gentle “lub-dub”—the heartbeat’s quiet rhythm—becomes more than just a sound. It becomes a living picture on a screen.

With each beat, the valves open and close in perfect harmony, sending tiny vibrations through the body. An echocardiograph captures these invisible movements, turning them into graceful images on a screen. What was once only heard is now seen—the heart’s chambers expanding and contracting, its strength revealed in every pulse.

Like a silent storyteller, the echocardiograph traces the journey of blood—flowing smoothly through the chambers, passing through each valve with purpose, and moving onward to sustain life.

THE INVENTION:

The story of the echocardiograph begins in 1953, when two visionary Swedish physicists—Inge Edler and Carl Hellmuth Hertz—first captured an image of the beating heart. What started as a breakthrough experiment soon became a window into life itself.

With the power of high-frequency sound waves—ultrasound, beyond 20,000 Hz—this remarkable “picture scope” allows us to see the human body’s most vital engine: the heart. Once hidden within the chest, its movements are now revealed in real time, each beat a testament to life’s rhythm.

Today, the echocardiograph stands as a gift to modern medicine. It empowers doctors to detect problems early, often before symptoms become severe, offering patients a chance at timely treatment and renewed hope.

From the very first step of identifying the cause of a symptom, the echocardiograph becomes the cardiologist’s trusted companion—confirming diagnoses, assessing cardiac function, monitoring ongoing conditions, and evaluating the overall cardiac state.

In its quiet precision, it does more than capture images. It listens, it reveals, and it safeguards the rhythm of life.

WHAT DOES THE ECHOCARDIOGRAPH FIND?

The noninvasive and smart application of this machine detects blood clots, congenital heart disease, cardiomyopathy, heart tumors, pericarditis,

thoracic aortic aneurysm, and multiple valve diseases. It is, in essence, a window into the heart’s structure and function—revealing what cannot be seen or heard alone.

THE WORKING:

The echocardiograph uses ultrasound technology to create an image of the heart.

Step 1: The transducer sends sound waves to the chest. These waves bounce off the heart tissue, producing echoes (reflections of sound).

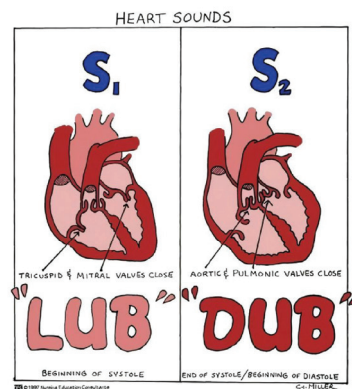
Step 2: The transducers capture these returning sound waves, called echoes, and send the signals to the computer.

Step 3: The computer software uses these echoes to create a moving picture of the heart on the screen. Because this entire process relies on ultrasound—sound waves beyond human hearing—it avoids the risks associated with radiation. Unlike X-rays or nuclear imaging, echocardiography is safe, painless, and repeatable, making it ideal for continuous monitoring and early diagnosis.

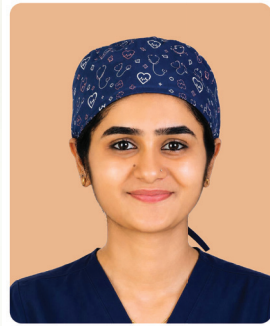
Your trust drives our mission:

We, as technicians, doctors, and your assistants, are committed to ensuring your comfort. With confidence and care, we bring to view the image of the heart—the tireless pump that beats with resilience, love, and faith.

Through every scan, we witness its strength and purpose, journeying miles within you, carrying life forward—reminding each of us of the very best that resides in every heartbeat.



“BEYOND THE SCALPEL: WHY EVH IS THE FUTURE OF INFECTION-FREE CARDIAC REVASCULARIZATION”



Ms. Lakshmipriya Manoj
Physician Assistant
Department of Cardiothoracic and
Vascular Surgery

Background

Traditional open vein harvesting (OVH) for Coronary Artery Bypass Grafting is frequently associated with significant leg wound morbidity, with infection rates reported as high as 25% in literature. This study evaluates the efficacy of Endoscopic Vein Harvesting (EVH) in reducing surgical site infections among patients with significant metabolic and systemic comorbidities.

Methods

A retrospective observational study was conducted on 336 consecutive patients who underwent CABG with EVH at a single tertiary center. Comprehensive data on demographics, comorbidities (Diabetes Mellitus, Hypertension, Chronic Kidney Disease, Dyslipidemia), surgical urgency, and post-operative wound infections were analyzed. Patients were followed up at 1 month and 3 months post-surgery to assess harvest-site recovery and delayed complications.

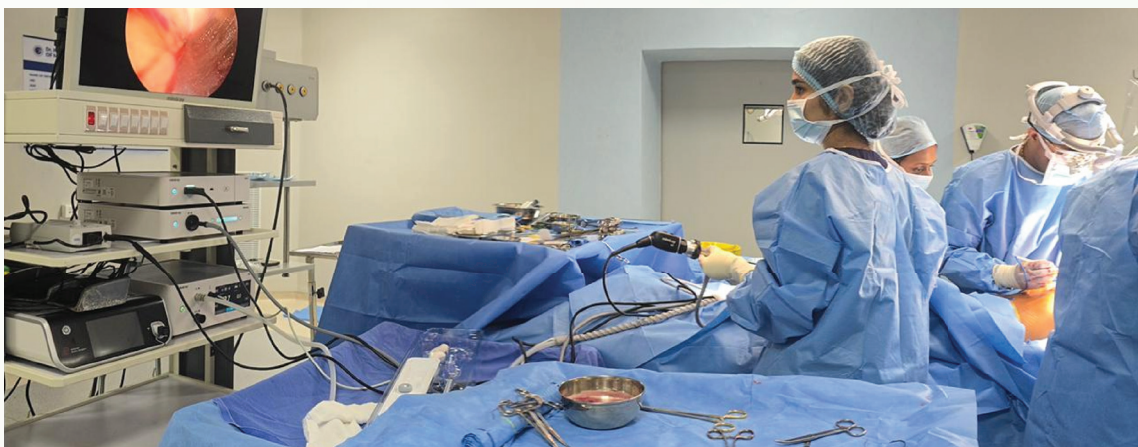
Results

The cohort (mean age 62.7 years; 82.7% male) exhibited a significant comorbidity burden: 78.6%

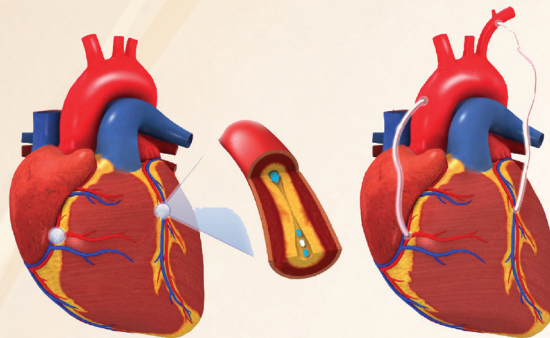
(n=264) had Diabetes Mellitus, 73.8% (n=248) had Hypertension, and 34.5% (n=116) had Chronic Kidney Disease. Despite this high-risk profile, the incidence of harvest-site wound infection was 0.00% (0/336). At the 1-month follow-up, 92.3% of patients reported no complaints, while 7.7% reported minor post-operative pain. By the 3-month follow-up, 100% of patients achieved complete recovery with nil complaints. No delayed infections or wound dehiscence were observed, regardless of surgical urgency (90.5% elective vs. 9.5% urgent/emergency).

Conclusion

Endoscopic vein harvesting is a highly safe and effective technique that virtually eliminates harvest-site infections. Its ability to maintain a zero-infection rate, even in patients with significant risk factors like Diabetes and CKD, suggests that EVH should be considered the gold standard for conduit harvesting in modern cardiac surgery to minimize patient morbidity and enhance post-operative recovery.



YOUNG AGE CABG: A RESEARCH DEPARTMENT OBSERVATION



Research & Development Department, Dr. K M Cherian Institute of Medical Sciences

Coronary Artery Bypass Grafting (CABG) is often considered a surgery for older adults. However, a review of CABG surgeries performed at Dr. K M Cherian Institute of Medical Sciences between April 2025 and March 2026 revealed an important finding: **9 patients below the age of 45 years underwent CABG surgery.**

Among these patients:

Youngest patient: **25 years**

Age range: **25–44 years**

Male patients: **6 (66.7%)**

Female patients: **3 (33.3%)**

Several patients required **4 to 6 grafts**, indicating advanced coronary artery disease despite their young age.

What Does This Finding Suggest?

The occurrence of severe coronary artery disease requiring bypass surgery at a young age is a matter of concern. Traditionally, heart disease was more common among older adults, but recent trends indicate that younger individuals are increasingly being affected.

Why Can Heart Disease Occur at a Younger Age?

Several factors may contribute to the development of coronary artery disease in young adults:

Diabetes mellitus

High blood pressure

Elevated cholesterol levels

Smoking and tobacco use

Obesity and sedentary lifestyle

Unhealthy dietary habits

Chronic stress

Family history of heart disease

When these risk factors are present, plaque can build up in the coronary arteries earlier in life, leading to significant blockages that may require surgical intervention.

What Can Be Done?

Prevention and early detection are essential.

Recommended measures include:

Regular health screening for blood pressure, blood sugar, and cholesterol.

Maintaining a healthy diet and body weight.

Engaging in regular physical activity.

Avoiding smoking and tobacco products.

Managing stress effectively.

Seeking timely medical evaluation for symptoms such as chest pain, breathlessness, or unexplained fatigue.

Early screening for individuals with a strong family history of heart disease.

Conclusion

The identification of nine CABG patients below 45 years of age in our hospital within one year highlights the growing burden of premature coronary artery disease. This finding emphasizes the importance of increasing awareness, promoting healthy lifestyles, and encouraging early cardiovascular screening among younger adults to reduce the risk of serious heart disease.

Editorial Team

Dr. Sarah J. Easaw

MBBS, MD, FACP
Medical Director,
Oncology Centre at
Dr. KMC Hospital

Dr. Rebecca John

Chief Operating Officer

Dr. Devarajan K. A

MBBS, MD, DM
Consultant Cardiologist

Nebu Sam John

Marketing Manager

Dr. Arun Thomas

MBBS, MD, Fellowship in Neonatology
(IAP & RCPCH)
Consultant Neonatologist

Meera Mariyam Skariah

Coordinator, Research and Development

EVENTS



20th February 2026

Dr. KM Cherian Institute of Medical Sciences, Chengannur, in association with the Senior Citizens Fellowship of the Changanur-Mavelikkara Diocese of Mar Thoma Syrian Church, organized the health seminar for senior citizens at Mar Thoma Parish, Karthikappally. Dr. Vishnu P. S. (Consultant Neurosurgeon, Dr. KMC Hospital) and Dr. Arun Babu (Consultant Oral & Maxillofacial Surgeon, Dr. KMC Hospital) delivered the awareness sessions.



3rd March 2026

Dr. Sarah J. Easaw (Medical Director & Consultant Oncologist, Oncology Centre, Dr. KM Cherian Institute of Medical Sciences) delivered a seminar on the topic "Changing Lifestyles in Kerala and the Health Issues Arising from Them" at the Health Arts Festival "Aarogam Aanandam", organized under the leadership of Health Minister Smt. Veena George. The event was inaugurated by Health Minister Smt. Veena George.



8th March 2026

Dr. Sannia Salim (Consultant Plastic, Reconstructive & Aesthetic Surgeon, Dr. KMC Hospital), was honoured with the 'Shero's Award 2026' by JCI Pathanamthitta on International Women's Day.



12th March 2026

On World Kidney Day, the Department of Transfusion Medicine & Nephrology at Dr. KM Cherian Institute of Medical Sciences organized the Blood Donation Drive.



14th March 2026

As part of the International Women's Day celebrations, Dr. KM Cherian Institute of Medical Sciences, Chengannur, in association with Mathru Vedi-Pithru Vedi of St. Mary's Unit, organized the cancer awareness seminar at St. George U.P. School, Eravuchira, Kottayam. The seminar was led by Dr. Sarah J. Easaw (Medical Director & Consultant Oncologist, Oncology Centre, Dr. KMC Hospital).



17th March 2026

The Advanced Centre for Liver Disease and Transplantation at Dr. KM Cherian Institute of Medical Sciences was inaugurated by the Hon'ble Minister of State for Health, Government of Maldives, Mr. Mohamed Mahir. The approval certificate from the Government of Kerala for conducting liver, pancreas and small intestine transplants was received by him from Dr. Noble Gracious (Executive Director, Kerala State Organ and Tissue Transplant Organization).

EVENTS



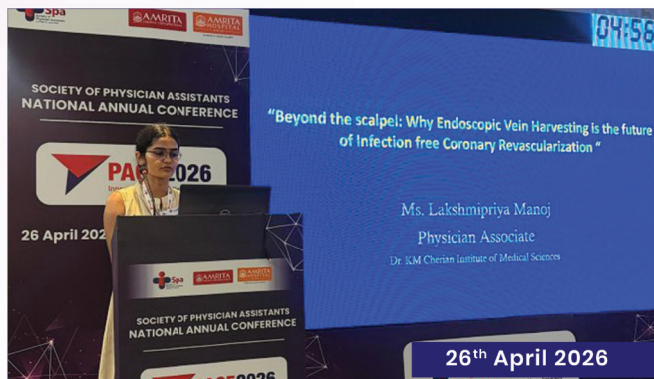
6th April 2026

The Advanced Neuro and Spine Centre at the Neurosurgery Department of Dr. KM Cheriaan Institute of Medical Sciences, was inaugurated by Dr. Ciza Thomas, Vice Chancellor of APJ Abdul Kalam Technological University. Standing Nearby: Rev. Fr. Dr. Alexander Koodarathil (Managing Director, Dr. KMC Hospital), Dr. M. V. Pillai (Advisory Board Chairman, Oncology Centre, Dr. KMC Hospital), Dr. Geevarghese K. Mathew (Medical Superintendent and Cardiothoracic and Vascular Surgeon), Dr. Vishnu P. S (Consultant Neurosurgeon), Dr. Sreenath P. R (Consultant Neurosurgeon) and Dr. Melvin K. Ninan (Consultant Neuro Anaesthetist)



7th April 2026

Dr. KM Cheriaan Institute of Medical Sciences organized an awareness session at Chengannur Railway Station on World Health Day. The session was led by Dr. Jibi Zachariah.



26th April 2026

Scientific Session by Ms. Lakshmi Priya Manoj (Physician Assistant, Department of Cardiothoracic and Vascular Surgery, Dr. KMC Hospital, Chengannur) at the National Conference of the Society of Physician Assistants, Kochi



5th May 2026

Department of Orthopaedics at Dr. KM Cheriaan Institute of Medical Sciences, Chengannur, organized the Orientation & Training Program for Fitness Trainers under MEDIFIT Clinic (Kerala's first clinically guided fitness initiative). The program was aimed at upgrading the skills of fitness professionals and enabling them to provide safer, science based training to their clients.



7th May 2026

Dr. KM Cheriaan Institute of Medical Sciences, Chengannur, organized the ATPP (Accident Treatment Program with Public Support) training for the Idukki Police Department at Thodupuzha. The function was inaugurated by Shri. Byju. P. M (DySP, Thodupuzha) and was attended by Shri. Basheer (Traffic SI, Thodupuzha), Shri. Biju. V. A (PRO, Thodupuzha DySP Office). The ATPP sessions were led by Dr. Arun Raj S. L (Consultant Emergency Medicine, Dr. KMC Hospital) and Dr. Ruben John (Consultant Psychiatrist, Dr. KMC Hospital). 70 police officers from the Idukki District Police Department underwent the ATPP training program.



7th May 2026

"Intraventricular Fibrinolysis: A Novel treatment strategy" – Scientific session by Dr. Vishnu P. S (Consultant Neurosurgeon, Dr. KM Cheriaan Institute of Medical Sciences, Chengannur) at Central Travancore Neuro Club Monthly Meet, Thiruvalla

EVENTS



10th May 2026

Department of Neonatology at Dr. KM Cherian Institute of Medical Sciences, in association with the National Neonatology Forum, organized the Nationwide NRP Day – an initiative for mass training in the Basic Neonatal Resuscitation Program (NRP) in collaboration with the Gov. of India, IAP, UNICEF, FOGSI and TNAI.



10th May 2026

Dr. Winnie Elizabeth Jose (Consultant Pulmonologist, Dr. KMC Hospital) participated in the panel discussion on 'Severe Asthma' at the Apollo Thoracic Interventions Conference (ATICON 2026), held at Apollo Hospitals, Chennai.



12th May 2026

Scientific sessions on "Oncoreconstruction: Where Function, Aesthetics and Plastic Surgery Finesse Unite" by Dr. Sannia Salim (Consultant Plastic Reconstructive & Aesthetic Surgeon, Dr. KMC hospital) and "Minimally Invasive is No Longer the Future – It is the Present – Practical Applications of Interventional Radiology in Everyday Practice" by Dr. Jaimi Abraham (Consultant Interventional Radiologist, Dr. KMC hospital, Chengannur)



16th May 2026

Nurses Day Celebration 2026 was Inaugurated by Cine Actress Prayaga Martin.



WINC 23rd May 2026

"The Nocturnal Dilemma: Restless Leg Syndrome in CKD revisited" Scientific session by Dr. Varsha Vipin, (Consultant Nephrologist Dr. KMC hospital) at WINCON 2026 held at Kumarakom.



26th May 2026

Department of Orthopaedics at Dr. KM Cherian Institute of Medical Sciences launched Medifit Clinic inaugurated by film actor Shri. Narain, Standing Nearby: Rev. Fr. Dr. Alexander Koodarathil (Managing Director, Dr. KMC Hospital) Dr. Krishnakumar Mahadevan (Sr. Consultant Orthopaedic Surgeon, Dr. KMC Hospital), Dr. Varghese Thomas (Consultant Orthopaedic Surgeon, Dr. KMC Hospital), Dr. Jobin Alex Mohan (Consultant Orthopaedic Surgeon, Dr. KMC Hospital), Dr. Geevarghese K. Mathew (Medical Superintendent & Consultant Cardiothoracic and Vascular Surgeon) and Dr. Naveen Pillai (Sr. Consultant Anaesthetist)

EVENTS



30th May 2026

Dr. KM Cherian Institute of Medical Sciences organized a Doctors' Panel Discussion on World Tobacco Day, centered around the theme "Unmasking the Appeal – Countering Nicotine and Tobacco Addiction." The panel discussion was led by Dr. Winnie Elizabeth Jose (Consultant Pulmonologist), Dr. Preethy V. Varghese (Consultant Neurologist), Dr. Ruben John (Consultant Psychiatrist), Dr. Nidhun V. Ashok (Consultant Oncologist) and Dr. Jibi Zachariah (Cardiology).



31st May 2026

"NEC: Early Recognition, Risk Stratification and Time-Critical Decision Making" – A scientific session delivered by Dr. Cherian Jerin Oommen (Consultant Pediatric Surgeon, Dr. KMC Hospital) at IMA Mavelikkara.



13th June 2026

In observance of World Blood Donor Day 2026, Department of Transfusion Medicine at Dr. KM Cherian Institute of Medical Sciences, Chengannur, organized an awareness session and voluntary blood donation camp. The sessions were led by Dr. Junith Thomas (Consultant Transfusion Medicine, Dr. KMC Hospital) Dr. Sarah J. Easaw (Medical Director and Consultant Oncologist, Oncology Centre, Dr. KMC Hospital) Dr. Sunil Agarwal (Consultant Cardiothoracic & Vascular Surgeon)



26th June 2026

Scientific sessions on "Beyond Tumor Removal: Restoring Form and Function Through Oncoreconstruction" by Dr. Sannia Salim (Consultant Plastic Reconstructive & Aesthetic Surgeon, Dr. KMC Hospital, Chengannur) and "Bench to Bedside: Evolving Landscape of Head & Neck Malignancy in a Tertiary Setting" by Dr. Gurukeerthi B. (Consultant in Head & Neck Surgical Oncology, Dr. KMC Hospital,) at IMA Pathanapuram.



26th June 2026

Department of Ophthalmology at Dr. KM Cherian Institute of Medical Sciences, Chengannur Launches 'Nermizhi – Cataract Free Kerala' Initiative. Inaugurated by Rev. Fr. Dr. Alexander Koodarathil. Standing Nearby: Dr. Gayathri. A (Consultant Ophthalmologist), Dr. Rebecca John (Chief Operating Officer), Dr. Muhamed A.V (Consultant Ophthalmologist) Dr. Kiran Balagopal (Consultant Ophthalmologist)



30th June 2026

Dr. KM Cherian Institute of Medical Sciences organized "A Breath of Fresh Air," – Lung Cancer Symposium, on Tuesday, 30th June 2026, bringing together experts from Oncology, Pulmonology & Radiology, for insightful discussions on the early detection, diagnosis, and multidisciplinary management of lung cancer. Chief Guest ; Dr. Tinku Joseph (Professor & Head of Interventional Pulmonology, Amrita Hospital, Kochi)

Hearty Welcome



Dr. Gurukeerthi. B
MBBS, MS, MCh (Head & Neck surgery,
Tata Memorial Hospital, Mumbai)
**Consultant in Head &
Neck Surgical Oncology**



Dr. Sreelakshmi M. V.
MBBS, DNB (Emergency Medicine)
Consultant Emergency Medicine



Dr. Shalini M. Nair
MBBS, MD (Anaesthesiology),
DrNB (Critical Care)
Consultant Critical Care



Dr. Muhamed A.V
MBBS, DO, DNB
(Ophthalmology),
Fellowship in Cataract Surgery
Consultant Ophthalmologist



Dr. Nikash P. John
MBBS, MS (Orthopaedics),
Diploma in Orthopaedics
**Consultant Orthopaedics
Surgeon**



Dr. Gayathri. S
MBBS, Dip DNB (Family Medicine)
**Jr. Consultant Family
Medicine**



Dr. Arya Bindu Rajoo
MBBS, MS (OBG)
**Jr. Consultant
Gynaecologist**



Dr. Sethu Lekshmi P
MBBS, DCH, DNB Pediatrics, MRCPCH (UK),
Fellowship in Neonatology
**Consultant Neonatologist &
Pediatrician**



Dr. Gayathri. A
MBBS, MS, Fellowship in Medical Retina
Consultant Ophthalmologist



Dr. Anand P. Nair
MBBS, MS, DNB, MCh,
DrNB (Neurosurgery)
Consultant Neurosurgeon



Dr. Syama Viswanath
MBBS, DNB (Emergency Medicine)
Consultant Emergency Medicine



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Kallissery P.O., Chengannur, Alappuzha (Dist)

Kerala, India - 689124 ☎ 0479 236 3300/ 3456, 0479 356 3456