TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

e - Newsletter

Issue 5

On

Heart Disease in Pregnancy

9th September 2021

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

TNFOG Team

President
Dr Anjalakshi Chandrasekar

Secretary
Dr S Sampath Kumari

Treasurer
Dr Vijayalakshmi Gnanasekaran

Vice President
Dr Revathy Janakiraman

Joint Secretary
Dr Radha Madhavi

Joint Treasurer
Dr S Saravanakumar

EC Members

OGSSI
Dr N Palaniappan
Dr Vijayalakshmi Seshadri

Krishnagiri
Dr E Chitra
Dr Rekha Rajesh

Tirunelveli
Dr Sheeba Jeyaseelan
Dr Nirmala Vijayakumar

Nagercoil
Dr Mary Ann
Dr Mini Gopal

Karur
Dr R Uma
Dr Nithya

Namakkal
Dr Chandra Ponnusamy
Dr R Vairamani

Tiruvannamalai
Dr Radha Madhavi
Dr Alamelu

Salem
Dr Vidhya Prabhakar
Dr Saravanakumar

Viruthunagar
Dr Aarthi
Dr Vinothini

Vellore
Dr Elsy Thomas
Dr Preethi R N

Madurai
Dr S Sumathi
Dr Ambigai Meena

Dindigul
Dr J Amala Devi
Dr Saroja Veluchamy

Trichy
Dr T Ramani Devi
Dr Deepa Mukundan

Theni
Dr Shanthi Rani
Dr Vanitha Rukmani

Thoothukudi
Dr S Pethukani
Dr Archana Arulraj

Thanjavur
Dr Thendral
Dr Uma Brindha

Erode
Dr. Nancy
Dr. Purnima

Coimbatore
Dr Suma Natarajan
Dr T V chitra

President's Message

Dear Comrades

Warm greetings from me. I am very happy that we are coming out with the Newsletter on heart disease complicating pregnancy. 0.2% to 4% of pregnancies have complications with cardiac disease. It can go upto 8% if hypertensive disorders are included. Cardiac disease of pregnancy encompasses a broad spectrum of pathology. Cardiac disease during pregnancy may be exacerbation of pre-existing condition, unmasking of asymptomatic patients during pregnancy both congenital and Rheumatic heart disease or they may develop a new disease process that presents because of the complex hormonal changes and physiology of pregnancy like peripartum cardiomyopathy, Arrhythmias etc. Cardiac disease is a significant cause of morbidity and mortality present between to 4% of all pregnancies. With advances in diagnostic tools we are diagnosing more and more cardiac problems and maternal echo in the II trimester has become the standard of care nowadays. Although there is a significant risk involved in such pregnancies one can successfully treat the majority of these cases if early and careful follow up are a part of routine care. In this Marathon CME Prof. cardiologist Dr.Justin Paul is going to talk about the importance of Registry for cardiac disease. This Newsletter deals with diagnosis and management of congenital heart disease, Rheumatic heart disease complicating pregnancy and to improve the outcome and also on peripartum cardiomyopathy, how prompt recognition and treatment can give 100% salvage. The newsletter also contains an interesting update. I request all of you to recoup your knowledge and give better service to our mothers.

Enjoy reading it and stay safe.

Jai Hind

Best Wishes

Dr. Anjalakshi Chandrasekar

Founder President, TNFOG

Secretary's Message

TNFOG is conducting this Marathon CME to encourage juniors and an update on subject. Each month we take one topic as theme of the month. This month's theme is 'Heart disease complicating pregnancy'. Heart disease complicates 0.2 – 4% of all pregnancies in the western world. The UK Obstetric Surveillance System (UKOSS) study of acute mi in pregnancy estimated an incidence of 0.7 cases per 100,000 maternities.

Most of the heart problems in women are diagnosed only during pregnancy'. An Indian study states that the prevalence of heart disease among pregnant women was found to be 2.32%. If not diagnosed earlier and treated, cardiac problems can lead to dreaded complication. Such patients are to be counselled about complications such as infection, cardiac failure during & after delivery. In order to reduce mortality, it is better to do echo as screening test for all antenatal patients during earlier weeks of pregnancy & help women to have safe deliveries with all necessary drugs & equipment. One child norm is safe for heart disease patients. New guidelines recommend that pregnant women with heart disease should give birth at no later than 40 weeks gestation.

Happy Vinayaga Chaturthi to All.

Best Wishes

Dr. S. Sampath Kumari

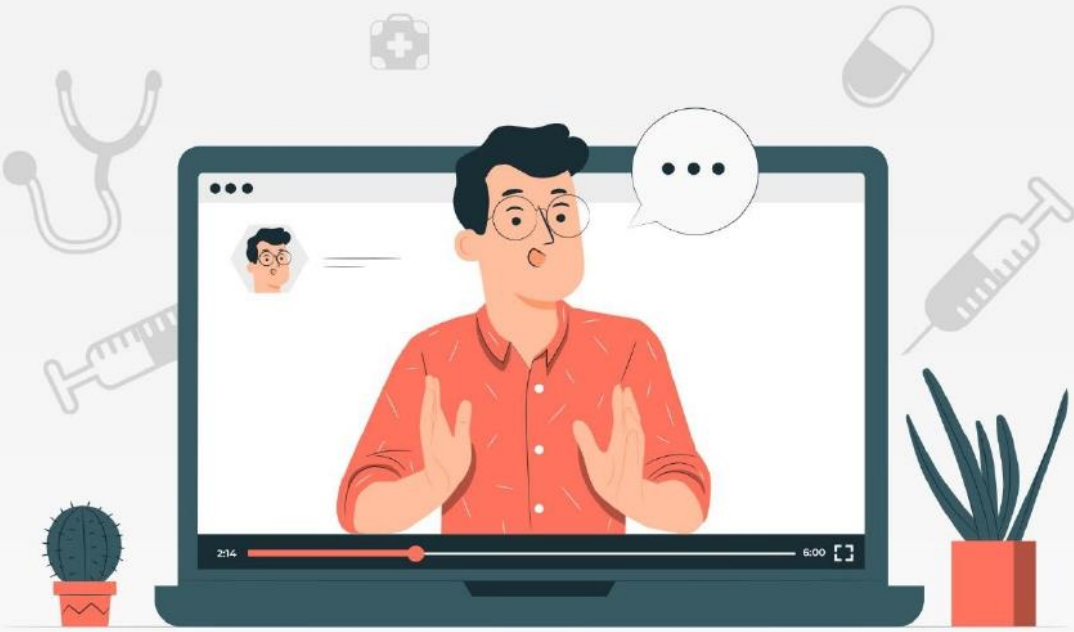
Founder Secretary, TNFOG

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

TNFOG Plans to conduct TWO CME Program Every Month

1. Marathon CME 2. Magalir Nalam

MARATHON CME??
THIS POINTS TO TWO THINGS



1. The YUVA OGCIAN Competition

Yes, every month 2nd Friday CME will have a session with 2 YUVA speakers, Consultants less than 35 years.

The session will be judged by the same judges and at the end of the year,
First, Second and Third prize will be awarded to the best speakers at the Annual conference.

All societies gear up and suggest one YUVA speaker of your society.

2. There is a question at the end of every session in the CME. The first Delegate who answers the question will be awarded a prize. This will continue in all the CMEs.

BE READY TO WIN THE PRIZE

TNFOG MARATHON CME ON

HEART DISEASE IN PREGNANCY

Date: 09.09.2021 (Thursday) | **Time: 4.30 - 7.15 PM**

Dr. Anjalakshi Chandrasekar
President , TNFOG

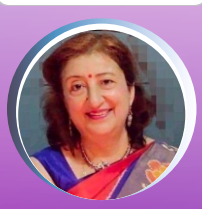
Dr. S. Sampath Kumari
Secretary, TNFOG

Dr. Vijayalakshmi Gnanasekaran
Treasurer, TNFOG

Chief Guest

Dr. Alka Kriplani

Guest of Honour



Dr. Ranjana Khanna

Session I

Judges

Dr. Nidhi Sharma

Dr. Vijayalakshmi Kandasamy

Speakers

Dr. T. Rajalakshmi

Dr. Pradeepa

Session II

Chiarpersons

Dr. M. Chandra Ponnusamy

Dr. T.S. Meena

Speakers

Dr. Sumathi Senthil Kumar

Dr. G. Justin Paul

Session III

Moderator

Dr. R. Premalatha

Panelists

Dr. R. Deepa Thangamani

Dr. M. Vijaya Lakshmi

Dr. C. Sumathi

Dr. Thennarasi

Dr. Aarti Dargarh

Dr. A. Anita Thamaraiselvi

TNFOG MARATHON CME ON

HEART DISEASE IN PREGNANCY

Date: 09.09.2021 (Thursday) | **Time: 4.30 - 7.15 PM**

1 ICOG Credit Point Granted

Scientific Programme

DURATION	TOPIC	SPEAKERS
04.30 - 05.00 PM	**INAUGURATION**	
	Introduction	Dr. S. Sampath Kumari
	Inauguration	Tamil Thai Vazhthu & Kuthu Villaku
	Welcome Address	Dr. Anjalakshi Chandrasekar
	Chief Guest Address	Dr. Alka Kriplani
	Guest of Honour Address	Dr. Ranjana Khanna
	Release of e-Newsletter (Issue 5) on "Heart Disease in Pregnancy"	
SESSION I - YUVA SESSION Chairpersons : Dr. Vijayalakshmi Kandasamy & Dr. Nidhi Sharma		
05.00 - 05.15 PM	Pathophysiology of Cardiovascular system in normal & heart disease in pregnancy	Dr. T. Rajalakshmi
05.15 - 05.30 PM	Anticoagulants in heart disease in pregnancy	Dr. Pradeepa
	Q & A	
SESSION II Chairpersons: Dr. M. Chandra Ponnusamy & Dr. T. S. Meena		
05.35 - 05.55 PM	Update of heart disease in pregnancy	Dr. Sumathi Senthilkumar
05.55 - 06.10 PM	Registry of heart disease in pregnancy	Dr. G. Justin Paul
	Q & A	
SESSION III - PANEL DISCUSSION Moderator: Dr. R. Premalatha		
06.15 - 07.15 PM	**Heart Disease in Pregnancy - Case Scenarios**	**Panelists**
		Dr. R. Deepa Thangamani
		Dr. M. Vijayalakshmi
		Dr. C. Sumathi
		Dr. Thennarasi
		Dr. Aarti Dargarh
		Dr. A. Anitha Thamaraiselvi
	Q & A	
07.15 PM	Vote of Thanks	Dr. Vijayalakshmi Gnanasekaran
Coordinator - A. Vanitha		

Coordinator

Dr. A. Vanitha

Vote of Thanks

Dr. Vijayalakshmi Gnanasekaran

After Each Session, Answer the 'Question' FIRST & GET EXCITING PRIZE!

For All Registrants, Certificate will be Provided

Click to Join!

zoom LIVE

We solicit your presence

An Educational Initiative Sponsored by

APCOD | egrich | TOTALIS

Table of Contents

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

Article: 1

Rheumatic Heart Disease in Pregnancy

Dr. Deepa Thangamani

MD OG., DNB OG., MRCOG., FRCOG.,

Consultant Apollo First med hospitals, Chennai

Introduction

Rheumatic heart disease remains the number one worldwide cause of maternal cardiac complications in pregnancy. Since the symptoms of rheumatic fever typically do not present until the fourth or fifth decade, the pathophysiological changes associated with pregnancy may cause as many as 25% of these women first experience symptoms during pregnancy. At childbearing age, valvular heart disease is often due to rheumatic heart disease, particularly in low–middle-income countries

Valvular heart disease due to rheumatic heart disease has declined in many developed nations, but it remains a prevalent cause of maternal cardiovascular morbidity and mortality in developing countries. Mitral stenosis is most common in rheumatic Heart disease followed by valvular (mitral and aortic) regurgitation lesions, aortic stenosis, pulmonary stenosis. Mechanical

prosthetic valves pose unique challenges in management of the pregnant patient given the requirement for anticoagulation.
Given the complexity of valvular heart disease in pregnancy, women with congenital and acquired heart disease should be managed with a multidisciplinary approach before and throughout pregnancy.

General Principle

Medical management of women before, during, and after pregnancy with unoperated and operated RHD is a challenge and requires a multidisciplinary team of physicians, cardiologists, obstetricians, anaesthesiologists, and sometimes cardiothoracic surgeons.

Unoperated RHD is most commonly diagnosed in pregnancy when the increase in cardiac output and drop in vascular resistance unmask moderate or severe valve lesions, and it contributes to maternal mortality (within 42 days after delivery) and late maternal death (up to 1 year postpartum). Therefore, all women with RHD have an increased risk of poor maternal and foetal outcomes, which increases further in the presence of left or right ventricular dysfunction, pulmonary hypertension, atrial fibrillation, and any signs of heart failure. Stenotic lesions are less well tolerated than regurgitant lesions and occasionally require interventions that include BMV, cardiothoracic surgery, or termination of pregnancy.

Appropriate preconception counselling, including advice on contraception, should be the goal but is unfortunately not the reality.

Women with mechanical valve replacement require anticoagulation throughout the pregnancy, which can include warfarin, unfractionated heparin, or low-molecular-weight heparin. Management of these women is complex, and maternal and foetal risk differ according to treatment regimen.

Mitral Stenosis

Mitral stenosis accounts for 90% of rheumatic heart disease in pregnancy. Particularly if undiagnosed, this may be dangerous in pregnancy. Women may have been previously treated with valvotomy or valvuloplasty, but stenosis can recur. Just because a woman is asymptomatic does not mean that she will tolerate pregnancy and delivery without complications. All patients with significant MS should be counselled against pregnancy and intervention should be considered pre-pregnancy, favouring percutaneous intervention, even if asymptomatic, and even more so if the valve area is <1.0 cm^2

Effect of pregnancy on mitral stenosis

Even if a woman is asymptomatic at the beginning of pregnancy, she can deteriorate rapidly and develop pulmonary oedema. This is usually precipitated by tachycardia. This may be as a result of intercurrent infection, exercise, pain, anxiety or secondary to a failure to adequately increase stroke volume.

Tachycardia is particularly dangerous in mitral stenosis since diastolic filling of the left ventricle (which is impaired in mitral stenosis) is further decreased and there is a consequent fall in stroke volume and a rise in left atrial pressure precipitating pulmonary oedema. Most women who develop complications do so in the late second or third trimester or peripartum period. Poor prognostic features for development of pulmonary oedema include

a) Severe mitral stenosis as assessed by valve area <1 cm^2
b) Presence of moderate to severe symptoms prior to pregnancy

For patients with moderate uncomplicated mitral stenosis, symptoms may peak at around 20–24 weeks gestation as cardiac output and intravascular volume peaks, and then stabilize. Patients with severe mitral stenosis remain at risk of heart failure and pulmonary oedema through the third trimester and into the puerperium

Management in pregnancy

β-blockers should be used to slow the heart rate and allow time for left atrial emptying. Atrial fibrillation should be treated aggressively with digoxin and β-blockers. Avoid injudicious intravenous fluid therapy. Avoid the supine and lithotomy positions. Pulmonary oedema should be treated with oxygen, diamorphine and diuretics.

In expert hands, balloon valvotomy and closed mitral valvotomy have very good results in pregnancy but are only suitable for non-calcified valves with minimal regurgitation. Suitability for valvuloplasty is usually assessed with transoesophageal echocardiography (TOE). Surgical valvotomy carries higher risks, with foetal mortality rates of 5%–15% for closed valvotomy and 15%–33% for open valvotomy. If women with severe mitral stenosis attend prior to pregnancy, they should be offered surgery (open/closed/balloon mitral valvotomy or valve replacement) before embarking upon pregnancy. Cardiac surgery should be considered in cases of unresponsive failure with pregnancy beyond 12 weeks. Best time of surgery is between 14 weeks and 18 weeks. Valve replacement, commissurotomy, balloon valvotomy can be carried out.

Atrial fibrillation is a complication. Digoxin, beta blockers and anticoagulants should be used.

Vaginal delivery is considered in women with mild mitral stenosis, and in patients with moderate mitral stenosis. Even in women with severe MS in whom symptoms are New York Heart Association (NYHA) Class I-II without pulmonary hypertension, vaginal delivery is considered. Caesarean section may be preferred in patients with severe mitral stenosis with NYHA Class III-IV symptoms, or who have pulmonary hypertension despite medical therapy

The greatest risk for an adverse cardiac event occurring was during labour and in the immediate post-partum period. Hence delivery was best carried out at a tertiary center with intensive care facilities and under the joint supervision of an obstetrician and cardiologist. Antibiotic prophylaxis against infective endocarditis was not recommended. Assisted vaginal delivery with instruments was preferred, in order to prevent maternal strain and exhaustion

Epidural analgesia was recommended for all patients undergoing vaginal delivery, as it prevented tachycardia without significant hemodynamic changes. This avoided the sudden increase in blood flow across the mitral valve and prevented sudden rise in left atrial pressure. Caesarean section only for patients with cardiac dysfunction, patient at risk of hemodynamic instability, pulmonary hypertension, uncontrolled arrhythmia, mechanical valve prosthesis, and patients with cyanosis. Regional anaesthesia, either epidural or spinal was safe in cardiac patients presenting for caesarean section.

Regurgitant valve disease

Systemic vasodilation and a fall in peripheral vascular resistance reduce after-load and therefore act to reduce regurgitation. Both mitral and aortic regurgitation are well tolerated in pregnancy, provided there is no significant left ventricular dysfunction. Women with heart failure can be safely treated with diuretics, digoxin and hydralazine and/or nitrates as vasodilators to 'offload' the left ventricle.

Mechanical Heart Valves

Management of anticoagulation in women with mechanical heart valves poses a unique challenge given the risk of valve thrombosis and risk of adverse foetal outcomes. Warfarin is associated with foetal birth defects, and studies have shown this effect may be pronounced when used during weeks 6-12. The foetal risk appears to be dose dependent, with observed foetal risk similar to low molecular weight heparin (LMWH) when the daily dose is ≤5 mg of warfarin. Although maternal risk of valve thrombosis is lowest with continued warfarin use throughout all three trimesters of pregnancy, foetal risk is optimized with the use of LMWH or low dose (≤5 mg daily) of warfarin. Thus, weighing risks and benefits of both maternal and foetal health, warfarin continuation is generally recommended when daily maternal dose is ≤5mg daily, whereas switching to therapeutic LMWH should be considered in patients who require higher warfarin dosing. Low-dose aspirin is recommended in mechanical and bio prosthetic valves in the second and third trimester. When LMWH is used for anticoagulation, meticulous monitoring of anti-Xa levels is required. Anti-Xa

levels should be measured 4-6 hours after a dose with a goal range of 0.8–1.2 U/ml .

Conclusion

The rheumatic heart disease continues to contribute to the maternal morbidity. Although maternal mortality has reduced, near miss cases, or those requiring intensive care are still high. The multidisciplinary management by cardiologists and obstetrician, proper preconception and antenatal care reduces the adverse events resulting in satisfactory maternal and foetal outcomes.

Reference:

1. Perrel C, Bonanno C, Valve disease in pregnancy. Semin Perinatol 2014;38(5):273-284
2. Mitchelson JB, Cleveland DE, Optimal management of a pregnant patient in Rheumatic Heart disease. Kans JMed,2018. Aug 30;11(3)80-82
3. Drife JO, Lewis G, Clutton-Brock T, editors. Why Mothers Die:The Sixth Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom 2000–2002. London: RCOG Press; 2004
4. Thorne SA. Pregnancy in heart disease. Heart 2004;90:450–6
5. Knight M, Nair M, Tuffnell D, Kenyon S, Shakespeare J, Brocklehurst P, Kurinczuk JJ, (Eds.) on behalf of MBRRACE-UK. Saving lives, improving mothers' care - surveillance of. maternal deaths in the UK 2012–14 and lessons learned to inform maternity care from. the uk and ireland confidential enquiries into maternal deaths and morbidity 2009–14. Oxford: National Perinatal Epidemiology Unit, University of Oxford; 201

Article: 2

Congenital heart disease (CHD) in pregnancy

Dr. Shobana Mahadevan

MD, FRCOG

Consultant Obstetrician& Gynecologist

Seethapathy clinic & hospital, Chennai

The incidence of congenital heart disease in pregnancy is increasing as women after corrective surgery are able to reach the childbearing age. As many of them would have fallen out of follow up with cardiac centres, it is important for every obstetrician to know the basic principles of preconception counselling and pregnancy care in order to have the optimal outcome in pregnancy.

Simple CHD lesions

1. Mild pulmonary valve stenosis
2. Small, uncomplicated ASD/VSD/PDA and successfully repaired ASD/VSD/PDA and anomalous pulmonary venous connection without important residua.
3. Marfan's syndrome with normal aortic root dimension

Complex CHD

1. Tetralogy of Fallot
2. Transposition of great vessels
3. Cyanotic heart disease with normal pulmonary pressures/ eisenmenger's syndrome

PDA, ASD and VSD contribute to 60% Of cases.

PDA

Most cases of PDA are corrected during childhood and have an outcome comparable to the normal population. The uncorrected ones are at risk of congestive heart failure

ASD

Commonest heart disease in pregnancy. Usually well tolerated but there is a risk of hypotension following blood loss during delivery. Other complications are SVT and paradoxical embolism.

VSD

Impact to the pregnancy depends on the size of the shunt. If there is reversal with Eisenmenger's phenomenon, there is worse outcome

Congenital aortic stenosis

Most cases are associated with a bicuspid aortic valve. Significant obstruction results if the aortic valve area is <1 cm^2 or if the mean gradient is severe (>50 mm Hg in the non-pregnant state). The risk of angina, hypertension, heart failure and sudden death increase with increasing severity of disease. Indicators of risk

include a failure to achieve a normal increase in blood pressure in response to exercise.

Coarctation of aorta

If uncorrected before pregnancy, the risks are angina, hypertension and cardiac failure. Aortic dissection and rupture can be fatal. This risk can be mitigated by blood pressure control and beta blockade. Documentation of the type of repair and pre-pregnancy MRI to identify any aneurysm or post-stenotic dilatation is necessary.

Marfan's syndrome

This is an autosomally inherited disorder and 80% of women will have cardiac involvement in the form of mitral valve prolapse, mitral regurgitation and aortic root dilatation. If the aortic root is > 4cm, there is 10% risk of aortic dissection and rupture in pregnancy and therefore should be offered termination in early pregnancy. Pre-pregnancy correction, regular monitoring with ECHO and beta blockers are the different strategies to improve outcome. Elective LSCS is indicated if there is progressive dilatation or if the root > 4.5cm

Cyanotic heart disease

Common causes encountered in pregnancy are pulmonary atresia and TOF. There is high risk of maternal and perinatal morbidity and mortality. The outcome is better if resting O2 saturation is > 85% and Hb < 18gm%.

Tetralogy of Fallot

The residual right ventricular failure and pulmonary hypertension are important considerations in operated women. Maternal hypoxia and

paradoxical embolism are the complications which can be reduced by thromboprophylaxis and admission and oxygen therapy.

Fontan circulation

This is an iatrogenic circulation that results after surgery for tricuspid atresia or transposition with pulmonary stenosis.

Prognostic indicators

Pulmonary hypertension

Functional class of NYHA

Cyanosis (arterial oxygen saturation <80%).

Transient ischaemic attacks or arrhythmias

Heart failure

Left heart obstruction (mitral valve area <2 cm^2, aortic valve area <1.5 cm^2, aortic valve gradient [mean non-pregnant] >30 mm Hg)

Left ventricular ejection fraction [LVEF] <40%)

Fetal risks

Spontaneous miscarriages 15-25%

Cardiac anomalies. Fetal ECHO is mandatory at 18-20 wks

FGR (4-8%)

IUFD (3%)

Preterm delivery (10-12% and20-60% in complex anomalies).

Neonatal death

Management of women with complex congenital heart disease

Preconception counselling

Age- appropriate counselling should be offered starting at the time sexual maturity with a view to assessing the maternal risk. The modified WHO classification is known to be one of the most reliable one. Contraception and termination are advised when there is significant risk.

Modified WHO classification of maternal cardiovascular risk

WHO risk category for severe morbidity/ maternal death	Maternal condition
1-Mild increase in morbidity/ no risk of mortality	Small PDA, mild pulmonary hypertension Corrected ASD, VSD, PDA, anomalous pulmonary drainage
2- mild risk of mortality/ moderate risk of morbidity	Uncomplicated (without PHT) unoperated ASD, VSD corrected TOF
2-3. moderate risk of morbidity/ mortality	Marfan syndrome without aortic dilation Aortic dilation < 45mm in bicuspid aortic valve Repaired coarctation
3. significant risk requiring intensive care with a cardiologist	Cyanotic heart disease Other complex CHD Fontan circulation Aortic dilatation 40-45mm in marfan's/ 45-50mm in BCAV
4. Extremely high risk. Pregnancy contraindicated/ TOP advised	Pulmonary arterial hypertension of any cause Aortic dilatation >45mm in Marfan's/ >50 mm in BCAV Severe LV dysfunction/ EF< 30%/ NYHA class III-IV

Preconception

Maternal cardiovascular risk assessment (WHO classification) by the team of cardiologist/ MFM consultant

Complete history and physical exam

Prior records reviewed

Baseline – ECG, ECHO, lab studies

Cardiopulmonary exercise tolerance and exercise class

Need for surgical repair assessed

Discontinuation of teratogenic drugs considered

Genetic referral with a hereditary cardiac lesion (family history, 22q deletion)

Contraception until cardiac fitness achieved

Early pregnancy assessment my multidisciplinary team

Maternal-fetal risk assessment

Termination if indicated

If fit to continue, care in tertiary centres / very low risk in local hospitals by a consultant with special interest in cardiac disease in pregnancy

Patient stable/ very low risk	**If pulmonary HT, NYHA III/IV, cyanosis**
Joint management by cardiologist/ MFM	Care in tertiary hospital
Decison about place of care/ delivery	Need for early hospitalisation
Routine frequency of antenatal visits	Frequent visits
Check symptoms, ECHO as per protocol	Surgical intervention in specific conditions

Fetal assessment

First trimester aneuploidy and preeclampsia screening as routine

Target scan with fetal ECHO

Growth assessment and doppler at 28, 32, 36 wks

Low risk		**High risk**
Written delivery plan in the records Vaginal delivery with epidural Caesarean section for obstetric indications Antibiotic prophylaxis as indicated		Multidisciplinary meet at viability to discuss labour and delivery Delivery date as clinically indicated Caesarean section may be indicated

Inherited cardiac anomalies

The risk of the fetus having a congenital heart defect is higher if the mother rather than the father has congenital heart disease. Overall, the risk is about 2%–5% (i.e.well over double the risk in the general population).

The level of risk depends on the specific lesion and is higher for left-sided outflow tract lesions. If the fetus is affected, it tends to have the same lesion.

In women with an ASD, the risk of an ASD in the fetus is about 5%–10%; for aortic stenosis, the risk is highest (18%–20%).

Marfan's syndrome has autosomal dominant inheritance.

Antenatal care

Individualised care should be provided by a multidisciplinary team (MDT) – cardiologist, maternal fetal medicine consultant, MFM midwife, social services, institutional ethics team. Except those with very low risk, all should be managed in a tertiary centre. The frequency of visits is increased for those with severe risk.

I trimester- routine obstetric care

II trimester- MDT meeting to plan care, delivery, need for procedures and surgery as indicated

III trimester- close monitoring for worsening of symptoms/ moving close to hospital/ delivery plan discussed

Obstetric complications

PTD management – caution with tocolytics/ terbutaline should be avoided

Preeclampsia is poorly tolerated in those with reduced cardiac output

Termination of pregnancy

Surgical termination is safe and the choice of anaesthesia will depend on the maternal status. Mifepristone and misoprostol are safe upto 7 wks but the unpredictable bleeding is not advisable in unstable patients. TOP after 20 weeks is associated with same risks as for delivery and should be avoided. Mid-trimester medical termination is associated with prolonged duration, pain and RPOC

Labour and delivery

Most women should be allowed to go to term. Induction of labour is done for obstetric reasons and if there is worsening in third trimester.
Elective induction in high-risk cases in daytime with CHD team being present
Lateral tilt to maintain the preload and avoid hypotension.
Opioid and epidural analgesia safe
CS for obstetric indications under GA or CSE- avoid hypotension
Maternal pushing increases O2 consumption and decreases venous return.
Prolonged second stage should be cut short by forceps delivery, particularly in those with obstructive lesions.
Elective caesarean section is indicated when pushing can be potentially fatal such as Marfan's syndrome, aortic aneurysm, severe left heart obstruction, unwell mother.

Postpartum

Immediately following delivery there is 60-80% increase in cardiac output due to autotransfusion of blood from the involuting uterus and this gradually decreases in the next 24 hrs and resolves in 6-8 wks. The risk of cardiac failure is highest at this time. Those with arrhythmias and those at risk or signs of failure should be managed in the ICU. Patients who have remained clinically stable throughout pregnancy and delivery may be transferred directly to postpartum units with instructions to monitor for cardiopulmonary symptoms. Early ambulation and continued use of support stockings may reduce the risk of thromboembolism.

Contraception

Combined pills are contraindicated when there is history of thrombosis, cyanosis and hepatic dysfunction. POPs as pills or implants are safe.

Cu IUCD has the risk of bacteremia and bleeding leading to anemia. LNG-IUS is safe.

Though both tubectomy and vasectomy may be advised, the latter should be discussed as the husband may outlive the wife with a severe cardiac condition.

Indications for antibiotics

Not generally recommended by AHA but in practice, given in most centres as the consequences of infective endocarditis can be devastating.

Indicated in mechanical valves, cyanotic heart conditions, Eisenmenger's syndrome and severe aortic stenosis.

Antibiotics are given 30 before delivery

Thromboprophylaxis

Early ambulation

TED stockings

LMWH for pulmonary hypertension, cyanosis, uncorrected TOF, Fontan's circulation

References:

1. Handbook of obstetric medicine, Fifth edition, Catherine Nelson-Piercy
2. Management of pregnancy with complex congenital heart disease: A scientific statement from the American Heart Association. MM Canobbio et al, Circulation,2017.
3. Congenital heart disease in pregnancy,Helen Wallis, Sara Thorne. A review, sAGE journals, 2006

Article: 3

Peripartum Cardiomyopathy

Dr. J.Karthiga Prabhu

Professor,

Department of Obstetrics and Gynaecology

SRM Medical College Hospital and Research Centre

Potheri

Case Scenario

A 28-year-old Primigravida was admitted in labour at 39 weeks. She delivered a 3.25 kg female baby by vaginal delivery. On postnatal 2 she developed breathlessness. On examination she was afebrile, tachycardic with features of pulmonary oedema. She does not give history of any heart disease. Echocardiography findings was consistent with DCM with ejection fraction (EF) of 27% and features of congestive cardiac failure.

What is your diagnosis?

Introduction:

Peripartum cardiomyopathy (PPCM) is a rare form of dilated cardiomyopathy of unknown origin, that is unique to the pregnant women of all reproductive ages. PPCM is defined as the development of cardiac failure between the last month of pregnancy and 5 months postpartum in the absence of recognizable heart disease. It affects previously healthy pregnant women with a low incidence of

0.1% of pregnancies but has a high morbidity and mortality rate ranging from 7% to 50%. The incidence of PPCM varies among populations; it is approximately 1 in 3000 in the United States compared with 1 in 300 in Haiti. In India a study from Manipal reported 1 case per 1374 livebirths.

Risk factors for PPCM

- multiparity,

- advanced maternal age,

- multifetal pregnancy

- pre-eclampsia and gestational hypertension,

- prolonged tocolytic use

- African American race

Pathophysiology:

The exact pathophysiology is still unknown. The disease has a multifactorial origin. A number of possible causes have been proposed including myocarditis, abnormal immune response to pregnancy, maladaptive response to the hemodynamic stresses of pregnancy, stress-activated cytokines, and elevated prolactin.

Myocarditis – Decreased immune response during pregnancy may allow for unchecked viral replication and thus a greater likelihood of myocarditis in the setting of a viral infection.

Abnormal hemodynamic response – During pregnancy, blood volume (preload) and cardiac output increase and afterload decreases. These changes result in a reversible hypertrophy of the left ventricle to meet the needs of the

mother and fetus. PPCM might be due to an exaggerated decrease in left ventricular function when these hemodynamic changes of pregnancy occur.

Abnormal immune response - During pregnancy fetal cells released into the maternal bloodstream are not rejected by the mother because of the natural immunosuppression that occurs during pregnancy. However, after delivery, women lose the increased immunity, and if fetal cells reside on cardiac tissue when the fetus is delivered, a pathological autoimmune response may result in PPCM after delivery.

Apoptosis and inflammation: Women with PPCM have been identified with an increased concentration of plasma inflammatory cytokines such as tumor necrosis factor, c-reactive protein and Fas/Apo-1, a plasma marker for apoptosis (programmed cell death). Levels of Fas/Apo-1 were higher in women with PPCM than in healthy volunteers.

Excessive prolactin excretion: Hilfiker *et al* proposed a new pathogenic mechanism for PPCM as a result of excessive prolactin production. An unbalanced peripartum oxidative stress leads to the proteolytic cleavage of the nursing hormone prolactin, with the resultant formation of a 16-kDa sub-form. This sub-form is a potent antiangiogenic, proapoptotic, and proinflammatory agent and affects the endothelium, cardiac vasculature, and cardiac myocyte function.

Selenium deficiencies and malnutrition: Deficiencies in selenium and other micronutrients were thought to play a role in the pathogenesis of PPCM.

Deficiencies of selenium increase cardiovascular susceptibility to viral infections, hypertension and hypocalcaemia.

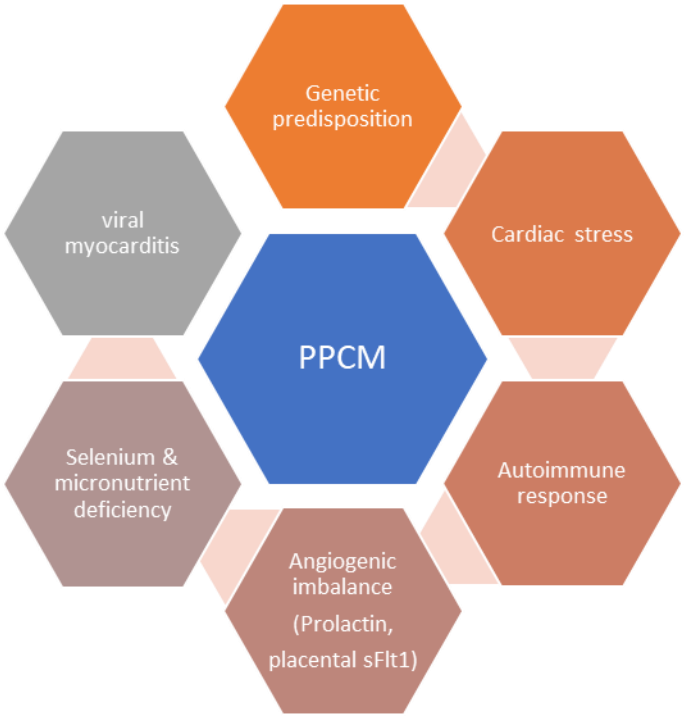


Figure 1 : Possible causes for PPCM

Clinical features:

Women usually present with common symptoms of pregnancy such as dyspnea on exertion, fatigue, edema, orthopnea and paroxysmal nocturnal dyspnoea, so the diagnosis of PPCM may be delayed or missed altogether. Late diagnosis has been associated with poorer outcomes, including persistent cardiac dysfunction and increased mortality. Women may present with heart failure, cardiogenic shock, arrhythmias, or stroke secondary to left ventricular thrombus.

Other types of cardiomyopathy will generally present in the second or early third trimester when the hemodynamic stresses are maximum. In contrast, majority of women with PPCM (75%) usually present in the postpartum period.

Diagnostic criteria for PPCM

1. Development of cardiac failure in the last month of pregnancy or within 5 months of delivery
2. Absence of an identifiable cause for the cardiac failure
3. Absence of recognizable heart disease before the last month of pregnancy
4. LV systolic dysfunction identified by classic echocardiographic criteria, such as ejection fraction less than 45% or fractional shortening less than 30%, or both

Echocardiogram, electrocardiogram (ECG), chest radiograph, cardiac MRI, and laboratory testing may all be useful in the diagnosis of PPCM. Echocardiography is noninvasive and allows serial evaluations in pregnant women. Echocardiographic findings in women with PPCM are consistent with the findings in heart failure such as decreased ejection fraction, global dilatation and thinned out cardiac walls. Cardiac magnetic resonance can be used as a complementary tool in the diagnosis of PPCM, Cardiac Magnetic Resonance Imaging can be used to measure global and segmental myocardial contraction, can help in characterizing the pathogenesis of the disease and can reveal inflammatory processes.

Many biomarker levels have been shown to be abnormal in women with PPCM. Markers of cardiac function such as N-terminal prohormone of brain natriuretic peptide (NT-proBNP), brain natriuretic peptide (BNP), and cardiac troponin are likely the most clinically useful.

Differential diagnosis of PPCM are myocardial infarction, amniotic fluid embolism, severe pre-eclampsia, pericarditis, pulmonary thrombo-embolism, myocarditis, sepsis, drug toxicity, and metabolic disorders.

Management

Management of PPCM is similar to usual treatment for other forms of dilated Cardiomyopathies except that angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists should not be used in antenatal women. The main aims in treating heart failure are to improve hemodynamic status, minimize signs and symptoms, and improve the long-term outcomes. Teamwork among obstetricians, cardiologists, neonatologist and anesthesiologists, is essential in the management of PPCM.

The main stay of treatment includes sodium restriction, diuretics, vasodilators and digoxin. Beta-blockers such as carvedilol have been shown to reduce mortality in dilated cardiomypothy. Anticoagulation with heparin should be initiated, if thrombus is noted on echocardiography. Anticoagulation is endorsed by the American Heart Association if EF is <30% during late pregnancy and up to 8 weeks postpartum. In addition, bromocriptine, a dopamine antagonist that inhibits prolactin secretion has been found to prevent the deterioration in the size of the left ventricle and systolic function when given in addition to standard heart failure therapy. But its role as a therapeutic agent is currently

experimental. If used, therapeutic anticoagulation is recommended, as it is prothrombotic and it suppresses lactation.

Temporary mechanical support should be considered early. Women with PPCM who have severe myocardial disease may benefit from a wearable or implantable cardiac defibrillator, left ventricular assist device (LVAD), mechanical circulatory support (MCS), and/or transplant.

Early delivery or termination of pregnancy should be considered in case of hemodynamic instability. Stable patients are delivered vaginally unless there are obstetric reasons for a cesarean section. Postpartum risk of decompensation should be anticipated. Given the intravascular fluid shifts associated with labor, delivery, and the immediate postpartum period, invasive blood pressure and central venous pressure monitoring are recommended. Neuraxial anesthesia appears ideally suited for these patients because it results in a beneficial decrease in both preload and afterload.

Progesterone-releasing subcutaneous implants or Mirena intrauterine devices are first-line choices of contraception and estrogen should be avoided.

Prognosis

The prognosis for women with PPCM depends on the normalization of left ventricular size and function within 6 months after delivery. Approximately 50% of affected women will continue to have symptoms of failure and cardiomegaly beyond 6months. These women should be advised against pregnancy as the incidence of recurrent disease is high. The usual causes of death in patients with PPCM are progressive heart failure, arrhythmia or thromboembolism. Factors associated with poor prognosis in PPCM include a

lower left ventricular ejection fraction at 6 months after delivery, larger left ventricular end-diastolic dimension, advanced maternal age, multiparty and African American descent.

Future pregnancies:

Appropriate counseling should be provided for patients regarding future pregnancies. If LV dysfunction persists, women must be counseled about poor maternal and fetal outcomes. Women who recover to EF >50% also may have an increased risk for HF in subsequent pregnancies. During subsequent pregnancies, women with PPCM should be closely followed with serial clinical assessments, echocardiograms, and B-type natriuretic peptide levels.

Key Points

1. Peripartum cardiomyopathy is a rare disease of unknown cause that affects women in the childbearing years, may recur, and is associated with a high mortality rate.
2. PPCM is a diagnosis of exclusion and should be based on classic echocardiographic criteria
3. Diagnosis of PPCM is challenging and requires vigilance. Diagnosing peripartum cardiomyopathy (PPCM) requires a high degree of suspicion, because presenting signs and symptoms tend to mimic those of normal pregnancy and the early postpartum period.
4. Careful assessment of risk factors in pregnant women could help in the prevention of PPCM.
5. Follow up measures are needed in order to predict the risk in future pregnancies.

6. Women with history of PPCM should be counseled about the risk in subsequent pregnancy.

References

1. Candice K. Silversides and Carole A. Warnes. Chapter: Pregnancy and Heart Disease . Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 90, 1780-1798
2. Mladen I. Vidovich. Chapter: Cardiovascular Disease. Chestnut's Obstetric Anesthesia, 41, 987-1032
3. Davis MB, Arany Z, McNamara DM, Goland S, Elkayam U. Peripartum Cardiomyopathy: JACC State-of-the-Art Review. J Am Coll Cardiol 2020;75:207-221.
4. Lucie Karafiatova, Marie Lazarova, Milos Taborsky. Peripartum cardiomyopathy – A case report and concise review. Cor et Vasa: Vol 59 (3) 2017, Pages e272-e276
5. Yamac H, Bultmann I, Sliwa K, Hilfiker-Kleiner D. Prolactin: a new therapeutic target in peripartum cardiomyopathy. Heart. 2010 Sep;96(17):1352-7.
6. Okeke T, Ezenyeaku C, Ikeako L. Peripartum cardiomyopathy. Ann Med Health Sci Res. 2013;3(3):313-319.

Article: 4

Update on heart disease in pregnancy

Dr. Sumathi Senthil kumar

DGO, DNB, FRCOG(UK), PGA in ART (Warwick University, UK)

Director & Consultant in OBG, SS Medical Centre, Salem

Maternal heart disease is a major threat to safe motherhood and women's long term cardiovascular health. Incidence of pregnancy in women with congenital heart disease and acquired heart disease is on the rise.

Rheumatic heart disease is common in India. Incidence of MI/IHD, Peripartum cardiomyopathy are increasing. Overall cardiac disease has risen due to advanced maternal age, obesity, survival of babies operated for CHD and awareness of investigating women with symptoms. Contributing factors for increasing morbidity, mortality due to Maternal Heart Disease are

1) Lack of pre -pregnancy CVS assessment and correct the defect or optimize the cardiac function.

2) Delay or failure to identify cardiovascular disease risk factors during Antenatal care.

3) Gaps in high- risk intrapartum care.

4) Delay or failure in recognition of CVS disease symptoms during puerperium

Symptoms and Signs of Heart Disorders in pregnancy:

SOB, Palpitations, Chest Pain, syncope, fatigue. Murmurs, jugular venous distention, tachycardia, dependent edema, mild cardiomegaly on chest X ray. Diastolic or presystolic murmurs are more specific for heart disorders. Heart failure can cause premature labour, arrhythmias and thrombosis.

Endocarditis Prophylaxis:

For pregnant patients with a structural heart disorder, indications and use of endocarditis prophylaxis for non-obstetric events are the same as those for nonpregnant patients. The American Heart Association guidelines do not recommend endocarditis prophylaxis for vaginal and cesarean deliveries because the rate of bacteremia is low.

Valvular Heart Disease:

During pregnancy, mild mitral or aortic regurgitation is usually easy to tolerate; stenosis is more difficult to tolerate and predisposes to maternal and fetal complications. Ideally valvular disorders are diagnosed and treated medically before conception. Surgical correction is often recommended for severe disorders.

Rheumatic heart disease is common in India. Mitral stenosis is the most common valvular disorder during pregnancy. MS is especially dangerous causing pulmonary edema and atrial fibrillation.

Anticoagulation and control of heart rate with beta-blockers, calcium channel blockers, or digoxin are necessary in atrial fibrillation. Pulmonary edema is treated with loop diuretics. Sometimes valvotomy Is needed. During labour, conduction anesthesia (eg, slow epidural infusion) is usually preferred in MS.

Aortic stenosis should be corrected before pregnancy if possible. Conduction anesthesia (epidural) should be avoided. Straining, which can suddenly reduce filling pressures and impair cardiac output, is discouraged during the 2nd stage of labour. Operative vaginal delivery is preferred. Cesarean delivery is done if indicated for obstetric reasons.

Mitral valve prolapse occurs more frequently in younger women and tends to be familial. MVP is usually an isolated abnormality that has no clinical consequences. Women with Mitral regurgitation in MVP generally tolerate pregnancy well.

Congenital heart disease (CHD):

Prevalence of this condition in pregnancy is about 0.8%. Deaths from congenital heart disease are fortunately uncommon. Most of them are asymptomatic. Risk is not increased during pregnancy except Eisenmenger syndrome, Pulmonary hypertension and Marfan syndrome. Congenital heart disease is one of the most common congenital abnormalities and the majority of those affected will survive to adulthood, in large part because of the development of effective corrective/palliative surgery over the last 30 years.

Acquired Heart Disease (Myocardial infarction, Ischaemic heart disease, and Aortic dissection)

Pregnancy itself raises the risk of acute myocardial infarction by three to four-fold, with the risk being 30 times higher for women over the age of 40 years compared with women aged less than 20 years. Risk factors: chronic hypertension, pre-eclampsia, diabetes, smoking, obesity and hyperlipidemia advanced maternal age, thrombocytosis and blood transfusion. Up to 1/13 women with a myocardial infarction in pregnancy will die. Incidence of maternal death is Increasing all over the world due to acquired heart disease and can occur in all stages of pregnancy. Systolic hypertension was a key factor in most of the deaths from aortic dissection, and this emphasizes the importance of blood pressure monitoring during pregnancy and prompt antihypertensive therapy if blood pressure becomes elevated. Aortic dissection (diagnosed by CT or MRI scan chest) is the most common serious complication of Marfan syndrome.

Peripartum cardiomyopathy:

The cause unknown in most cases. It usually presents in late pregnancy or early in the puerperium, but it can occur up to 6 months after delivery. Peripartum cardiomyopathy should be considered in any pregnant or puerperal woman who complains of increasing shortness of breath, especially on lying flat or at night. Recurrence is likely in subsequent pregnancies, particularly in patients with residual cardiac dysfunction; future pregnancies are therefore not recommended. Heart failure with no identifiable cause (eg, myocardial infarction, previous heart disorder) in puerperium, one should suspect cardiomyopathy. Risk factors: Age ≥ 30, Multiparity, Multiple pregnancy, Preeclampsia.

Prosthetic Valves:

Aspirin for bioprosthetic heart valves is safe in pregnancy and need not be adjusted. Anticoagulation (Heparin or LMWH) for mechanical heart valves should be adjusted. Warfarin must be discontinued during the 1st trimester due to the well documented risk of embryopathy.

Diagnosis of Heart Disorders in Pregnancy:

Auscultation of the chest for any murmur, crepitations should be done in the first visit for all pregnant women and that should be clearly documented. BP manually with a sphygmomanometer (mention the arm side and posture), PR (manual) to assess rhythm, rate, volume, SpO2 should be measured during each visit. Probing clinical history should be taken during each visit and auscultation of chest should be done for any murmur and crepitations if there is any symptom. There should be low threshold for even mild symptoms in pregnancy to investigate (ECG, Cardiologist opinion and Echo).

Antepartum care:

AN visit for known cardiac disease: once in 2 – 4 weeks till 20 weeks, once I 2 weeks till 24 weeks, then weekly once till delivery – continuity of care is very important.

Risk assessment should be done in each visit (probing clinical symptoms, BP, PR, SpO2 and auscultation of chest for increasing murmur or crepitations). Frequent prenatal visits, ample rest, avoidance of excessive weight gain and stress, treatment of anaemia are required. Regular cardiologist review is needed from early pregnancy itself. NT scan at 12 weeks and Fetal Echo at 22 weeks should be done. Genetic counselling should be offered for mother with CHD. Multidisciplinary team assessment (Obstetrician, Anaesthetist, Cardiologist, and Neonatologist) at 32-34 weeks to plan the care around the

time of delivery and mode of delivery should be done for all pregnant women with cardiac problem and should be documented in the notes clearly.

Intrapartum care: Delivery should be planned in a tertiary care centre where senior obstetrician, Cardiologist, Neonatologist and Anaesthetist with cardiology experience are available. Tertiary units should have high-dependency and intensive care units suitable for the care of pregnant women with significant heart disease.

The general principle of intrapartum management is to minimize cardiovascular stress.

- During labour, pain and anxiety are treated to minimize tachycardia – early epidural. In most cases this will be achieved by the use of early slow incremental epidural anaesthesia and assisted vaginal delivery.
- Back rest and O2 during labour
- Maternal monitoring: ECG, SpO2, Non-invasive BP, Arterial line BP, CVP,
- Caesarean section is usually necessary only for obstetric indications.

LSCS: Prophylactic compression sutures, Syntocinon 2 units over 10 minutes, Low dose Syntocinon infusion (8-12 mu / minute)

- Epidural can be given 12 hours after the prophylactic dose and 24 hours after therapeutic dose of LMWH / warfarin

Vaginal delivery:

- **1st stage**: TED's stockings, Epidural, Continuous CTG, prophylactic antibiotics and maternal monitoring
  Syntocinon augmentation of labour: double strength Syntocinon (half the rate to reduce total volume of fluids given)

- **2nd stage**: Avoid pushing, cut short 2 stage, assisted vaginal delivery by instruments

  3rd stage: Syntocinon 5 units IM or 2 units IV over 10 minutes and CCT, Syntocinon infusion (8-12 mu / minute). (5 units in 50ml at 7ml / hour or 10 units in 500ml at 36 ml / hour continue for 4 hours). **DO NOT GIVE ERGOMETRINE**

- **PPH:** Bimanual compression, Misoprostol 600 mcg P/R, Intrauterine balloon with antibiotic coverage, compression sutures. **AVOID PROSTADIN**

Strict I/O chart, consider central access or arterial monitoring

Preterm Labour: Atosiban is the first line, Avoid ritodrine or salbutamol

Pace maker: do not use Monopolar diathermy, Beware of pace maker in unusual places (eg. Abdominal wall), De-activate implantable defibrillators

Postpartum care: Includes close monitoring during immediate postpartum and cardiology follow up for several weeks. Should be monitored closely for 12-48 hours after delivery in high dependency unit. Anticoagulation should be given according to cardiologist advice.

Appropriate contraception advice should also be given.

Contraception: Natural method and Barrier method – high failure rate. Combined pills increase the risk of thrombosis. Progesterone only Pill (POP), Implant of Progestogen, Injectable Progestogen (Depo Provera) are safe to use. Mirena IUD (less chance of infection, bleeding and ectopic compared to copper IUCD). Male Sterilization (Vasectomy) should be encouraged. Female

Sterilization: Laparoscopy / Mini laparotomy.

Hysteroscopic insertion of Essure under Local or IV sedation

To reduce the Morbidity and Mortality: Pre-pregnancy /Premarital counselling: All women should be assessed for any cardiac disease before planning for pregnancy. Optimization of the cardiac status / corrective cardiac surgeries before planning for pregnancy to reduce the incidence of morbidity and mortality. Successful pregnancies can be achieved when cardiac complications are managed appropriately during pregnancy. In order to optimize maternal and neonatal outcomes, close collaboration between the Obstetrician, Anesthetist, neonatologist and the cardiologist is important.

Despite dramatic improvements in survival and quality of life for patients with severe congenital heart defects and other heart disorders pregnancy is inadvisable in certain conditions.

Contraindications for Pregnancy: Should be offered early therapeutic abortion.

Pulmonary arterial hypertension (pulmonary artery systolic pressure > 25 mm Hg) caused by any condition, including Eisenmenger syndrome

- Severe systemic ventricular dysfunction (EF <30%, NYHA III – IV)
- Coarctation of the aorta if uncorrected or if accompanied by an aneurysm
- Severe Aortic Dilation (aortic root diameter of > 45 mm in Marfan or other HTAD, Bicuspid aortic valve ascending aorta diameter >50 mm, Turner syndrome ASI >25mm/m2, TOF >50mm)
- Severe symptomatic aortic stenosis or severe mitral stenosis

A single ventricle and impaired systolic function
(whether treated with the Fontan procedure or not)
Fontan circulation with any complications

- Vascular Ehlers-Danlos
- Previous peripartum cardiomyopathy with any residual left ventricular dysfunction (ejection fraction < 30% or NYHA class III or IV heart failure)

Treatment of Heart Disease in Pregnancy Women with NYHA class III or IV status before they conceive, the disorder should be optimally treated medically and, if indicated (eg, if due to a valvular heart disorder), treated surgically

- Women with class III or IV heart failure or another high-risk disorder (listed above) may be advised to obtain an **early therapeutic abortion.**
- Some women with a heart disorder and poor cardiac function (class III or IV heart failure) require digoxin 0.25 mg orally once a day plus bed rest or limited activity, beginning at 20 weeks.
- Cardiac glycosides (eg, digoxin, digitoxin) cross the placenta, but neonates (and children) are relatively resistant to their toxicity.
- ACE inhibitors and angiotensin II receptor blockers (ARBs), are contraindicated because they may cause fetal renal damage.
- Aldosterone antagonists (spironolactone, eplerenone) should be avoided because they may cause feminization of a male fetus.
- Other treatments for heart failure (eg, nonthiazide diuretics, nitrates, inotropes) may be continued during pregnancy depending on disease severity and fetal risk, as determined by a cardiologist and a perinatologist.

Key Points

- Pregnancy may not be advisable for women with certain high-risk heart disorders.
- Optimization of the cardiac status / corrective cardiac surgeries before pregnancy to reduce the incidence of morbidity and mortality
- Treat heart failure and arrhythmias during pregnancy as for nonpregnant patients, except avoid certain drugs (eg, warfarin, ACE inhibitors, ARBs, aldosterone antagonists, thiazide diuretics, certain antiarrhythmics such as amiodarone).
- Treat most pregnant patients who have atrial fibrillation with standard or low molecular weight heparin.
- Indications for endocarditis prophylaxis for pregnant patients with a structural heart disorder are the same as those for other patients. Endocarditis prophylaxis is not needed for vaginal and caesarean deliveries
- Avoid Ergometrine, Prostadin, Ritodrine, salbutamol, High dose Syntocinon and monopolar diathermy and fluid overload.

References:

1. Tex Heart Inst J. 2011; 38(2): 151–153, Cardiac Disease in Pregnancy, Wayne J. Franklin, MD, FACC, Mary Kay Benton, RN, BSN, and Dhaval R. Parekh, MD
2. Heart Disorders in Pregnancy by Lara A. Friel, MD, PhD, University of Texas Health Medical School at Houston, McGovern Medical School Last full review/revision Jun 2021| Content last modified Jun 2021
3. ACOG Practice Bulletin, Clinical Management Guidelines for Obstetrician – gynaecologists, Number 212

Good Practice No.13 June 2011 Cardiac Disease and Pregnancy, RCOG

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

Article: 5

Pathophysiology of Cardiovascular system in Pregnancy & Heart Disease

Dr. T. Rajalakshmi

DNB(OG), DGO, F.MAS

Assistant Professor, Department of Obstetrics and Gynecology

Thoothukudi Medical College Hospital

Introduction

Around 1 to 3 % of all pregnancies are complicated by maternal cardiac disease, resulting in approximately one fifth of all maternal deaths. Knowledge of the normal physiology of pregnancy and risk associated with specific cardiac condition during pregnancy will help the obstetricians to diagnose and, in many cases, initiate the management of medical complications that may affect the pregnant women with heart disease.

Cardiac diseases are broadly divided into two categories namely congenital and acquired. There has been a decrease in rheumatic heart disease worldwide, but it continues to be leading cause in India accounting for 85 to 90% of cases.

Cardiovascular system during pregnancy

Anatomical Changes:

As the diaphragm is elevated, the heart is displaced to the left, upward and rotated on its long axis. So, the apex is moved upward and laterally to the

fourth intercostal space lateral to the mid clavicular line.
Some pregnant women have benign pericardial effusion.
Hence X-ray chest shows apparent cardiomegaly with straightening of left heart border.

There is spherical eccentric hypertrophy of heart and ventricular chamber size is increased with wall thickness maintained. ECG shows 15-degree left axis deviation, reduced mean PR interval, inverted or flat T waves, Q wave in lead DIII. On auscultation, we hear split first heart sound, loud third heart sound, ejection systolic murmer in pulmonary area, continuous murmer from mammary vessels.

Hemodynamic changes:

The hemodynamic changes begin early in the first trimester. The plasma volume start increasing by sixth week and approaches 50 % above baseline till end of second trimester and plateaus. Cardiac output start increasing as early as five weeks, reaches the peak at thirty weeks and plateaus till term. Up to 500 ml of blood is released into circulation with each uterine contraction at the time of delivery.

There is an abrupt increase in venous return after delivery of the baby because of auto transfer from uterus and relief of venacaval compression by uterus. All these changes increase the risk of pulmonary edema.

Picture: Changes in Cardiac Output, Stroke Volume,

Heart Rate during pregnancy

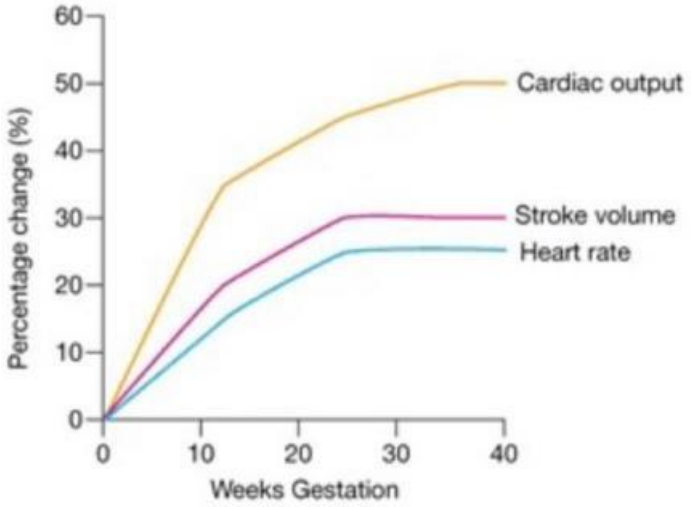


Table: Hemodynamic changes during pregnancy

Hemodynamic Parameter	**Changes during normal pregnancy**	**Change during labour & delivery**	**Change during postpartum**
Blood volume	Increases by 40-50%	Further increases	Decreases (auto diuresis)
Heart rate	Increases by 10-15 beats/min	Further increases	Decreases
Cardiac output	Increases by 30-50% above baseline due to • Increase in preload due to greater blood volume • Decrease in afterload due	Increases by additional 50% (Labour pains, uterine	Decreases

	to decrease in systemic vascular resistance • Increase in maternal heart rate by 10 to 15 beats/min	contraction, relief of caval compression after delivery and autotransfusion of blood from emptied and contracted uterus contribute to increased cardiac output)	
Blood Pressure	Decreases by 10 mm Hg in mid-trimester due to decrease in systemic vascular resistance	Increases	Decreases
Stroke volume	Increases in first and second trimesters; decreases in third trimester due to compression by gravid uterus	Increases (300 to 500 mL/contraction)	Decreases
Systemic vascular resistance	Decreases (smooth muscle relaxing effect of progesterone, nitrous oxide, prostaglandins)	Increases	Decreases

Supine Hypotensive Syndrome:

In supine position, large pregnant uterus compresses venous return from the

lower body decreasing preload and cardiac output causing significant arterial hypotension called as supine hypotension. Uterine blood flow also decreased. There is 20% increase in cardiac output when women turns from supine to left lateral position.

Effect of pregnancy on maternal cardiac disease:

Women with cardiac disease experience great hemodynamic alteration during the period of pregnancy predisposing to decompensation and congestive heart failure. There are special phases of pregnancy when the danger of cardiac decompensation is high as shown in the below table.

GESTATIONAL AGE	REASON
12 – 16 WEEKS	Hemodynamic changes of pregnancy begins
28 & 32 weeks (50% of those developing CHF belong to NYHA CLASS II / III at earlier GA)	Changes peak & cardiac demands are at maximum
During labour	300-500 ml of blood injected into circulation with each contraction
After delivery of baby and placenta	Auto transfusion & relief of compression of IVC
4-5 DAYS after delivery	Sudden death, Decreased peripheral resistance with R-L shunt, Pulmonary embolization

In all trimesters, Hypertension, Anaemia, Multiple pregnancy, thyrotoxicosis, Acute febrile illness precipitates heart failure.

Effect of cardiac disease on foetus:

The severity of the underlying lesion and degree of hypertension and hypoxia are the major determinants of pregnancy outcome. Foetal morbidity is due to preterm delivery and foetal growth restriction due to their relative inability to maintain uteroplacental circulation. Foetal death can occur in cyanotic heart disease.

Diagnosis of Heart Disease:

Some of the symptoms and signs that mimic heart disease are present in normal pregnancy also, but following symptoms and signs definitely suggest heart disease as shown in table below

Normal Pregnancy	Definite Heart Disease
Symptoms	
Dyspnoea	Severe dyspnoea (Orthopnoea, PND)
Fatigue	Nocturnal cough
Edema	Haemoptysis
Heart Burn	Severe chest pain
Palpitation	Syncope
Signs	
Neck veins pulsation	Neck veins distension
Systolic murmur < Gr. III	Systolic murmur > Gr. III, Diastolic murmur
Displaced apex beat	Definite cardiomegaly
Third heart sound	Loud P2, Wide split S2 Fourth H. sound
Sinus tachycardia	Persistent arrhythmia Cyanosis, Clubbing

Rheumatic Heart Disease

Mitral stenosis:

Mitral stenosis is the most common rheumatic heart disease. The contracted valve impedes blood flow from the left atrium to the ventricle. The normal valve area is 4-6 cm^2 and when stenosis narrows to < 2.5 cm^2, symptoms usually develop. Moderate stenosis is 1-1.5cm^2, severe stenosis is <1.5cm^2.

There is left atrial outflow obstruction and the resting pressure gradient across the mitral valve raises. With more severe stenosis, left atrium dilates, left atrial pressure is chronically elevated leading to increased pulmonary venous and capillary pressure and pulmonary hypertension. Dyspnoea occurs due to reduced pulmonary compliance and hemoptysis occurs due to pulmonary congestion.

Atrial dilatation makes it vulnerable to arrythmias and atrial fibrillation can occur. With stagnation of blood and atrial fibrillation, thrombus formation may occur with embolic complication. As stroke volume is fixed, Pregnancy alternation of increased cardiac output, tachycardia, fluid retention predisposes to pulmonary edema and heart failure. Prevention of anemia, infection, PE is of concern.

Mitral Regurgitation:

Acute MR is caused by chordae tendinae rupture, papillary muscle infarction. This causes flash pulmonary edema and life-threatening cardiac decompensation. Chronic MR is due to myxomatous degeneration or rheumatic heart disease. Due to regurgitation, there is left atrium dilatation and eccentric hypertrophy. During pregnancy, lowered systemic vascular

resistance improves the forward flow and regurgitation is decreased. Hence MR is well tolerated during pregnancy.

Mitral valve prolapse:

Mitral valve prolapse is due to myxomatous degeneration of the valve. Usually asymptomatic and diagnosed during routine examination or echo. Hypervolemia may even improve alignment of the valve. Few women have symptoms like anxiety, palpitation, atypical chest pain, dyspnoea with exertion and syncope.

Aortic stenosis:

It is either congenital or rheumatic in origin. Normal valve area is 3 to 4 cm2. The pressure gradient across the valve increase rapidly as the valve area is reduced to $< 2\ cm^2$. Mild to moderate is well tolerated but severe stenosis is life threatening.

Obstruction at the aortic valve leads to left ventricular overload, increase in left ventricular end diastolic pressure and fall in ejection fraction. Fixed cardiac output and progressive pressure overload leads to concentric left ventricular hypertrophy. Increase in cardiac output can only be met by increasing heart rate which shortens the diastole, so it decreases the time for coronary perfusion and ventricular filling. Thus cardiac, cerebral, uterine perfusion are decreased They develop chest pain, syncope, heart failure, sudden death.

During labour and delivery, they are managed on 'wet' side with IV fluids to avoid hypovolemia. Narcotic epidural analgesia is preferred, and care should

be taken to avoid hypotension. Abrupt drop in end diastolic volume may result in syncope, MI and sudden death

Aortic regurgitation:

Generally, well tolerated during pregnancy because the decrease in systemic vascular resistance will increase forward flow.

Pulmonary Stenosis:

It may be congenital or rheumatic in origin. Mild stenosis is well tolerated. Severe stenosis is associated with RA and RV enlargement and can precipitate right heart failure and arrythmias.

Women with Prosthetic heart Valves:

Bioprosthetic valves in the presence of a normally functioning left ventricle and absence of PHT are not associated with increased risk during pregnancy. Patient with mechanical prosthetic valves require lifelong anticoagulation. During pregnancy the risk of thrombosis increases further owing to hypercoagulation status. Pregnancy outcomes depend on the type of valve, number of replaced valves, functional capacity of heart following surgery.

European society of cardiology (ESC) guidelines recommend the use of low dose oral anticoagulation throughout pregnancy with strict weekly INR monitoring. Warfarin < 5 mg/day is rarely associated with warfarin embryopathy. In those who need higher dose, unfractionated heparin may be added between 6 and 12 weeks. LMWH can also be used but it should be discontinued 24 hours before delivery. Unfractionated heparin can be

substituted predelivery because it can be started and stopped rapidly.

Congenital Heart Disease

Pregnancy in women with surgically corrected congenital heart disease is now commonly encountered. Once the defect is repaired, the risk during pregnancy is minimal. Hemodynamic changes of pregnancy affect them. Fall in systemic vascular resistance increase the magnitude of left to right shunts. Risk of thromboembolism and infective endocarditis is increased. Arrythmias if present will worsen during pregnancy

Atrial Septal Defect (ASD):

Most ASD are asymptomatic until third or fourth decade. Pregnancy is well tolerated unless pulmonary hypertension is developed. With a potential to shunt from right to left, a paradoxical embolism, there is a entry of a venous thrombus through the septal defect and in to the systemic arterial circulation is possible and may cause embolic stroke. Compression stocking and prophylactic heparin have been recommended.

Ventricular Septic Defect (VSD):

These lesions usually close spontaneously or surgically corrected during childhood. If the defect is $< 1.25\ cm^2$, pulmonary hypertension and heart failure do not develop. If the defect size exceeds that of the aortic valve orifice, symptoms develop rapidly.

Adult with unrepaired large defects develop left ventricle failure and pulmonary hypertension and have a high incidence of bacterial endocarditis

and poor prognosis. The presence or absence of PHT decides the prognosis. If pulmonary arterial pressure reaches systemic levels, there is reversal of flow (Eisenmenger syndrome) with high maternal and foetal mortality.

Patent Ductus Arteriosus (PDA):

Rarely seen during pregnancy and outcome is determined by the presence or absence of pulmonary HT.

Cyanotic congenital heart disease:

They include corrected tetralogy of fallot, Transposition of great arteries, Ebstein Anamoly, double outlet right ventricle, single ventricle, Tricuspid atresia. 92% of them develop heart failure, stroke, SVT, endocarditis. The live birth rate is only 43% which is determined by arterial oxygen saturation (>90%) and haemoglobin concentration (<20 g/dl).

Outcome of pregnancy depend on the ejection fraction of the right ventricle, which should be atleast equal or larger than 40%. Women with corrected Tetralogy of Fallot tolerate pregnancy as long as ventricular function is good. Women with transposition of great arteries may develop heart failure and arrythmia.

Women with single ventricle are corrected with fontan operation where the ventricle is used to support systemic circulation but there is no pumping organ for pulmonary circulation. This makes it difficult to raise cardiac output and women develops CHF and arrythmia

Coarctation of Aorta:

It is rarely seen during pregnancy as majority are surgically corrected during childhood. The narrowing of the aorta is distal to the left subclavian artery resulting in isolated hypertension in the right arm.

Determining the arm leg pressure gradient which is abnormal when > 20 mm of Hg, assess the severity of coarctation. Pregnancy increases the risk of aortic dissection, ruptured aneurysm, infected endocarditis. Hypertension should be detected and controlled.

Marfan syndrome:

Inherited as autosomal dominant and hence mother should be informed of the 50% risk of transmission to their offspring. The main sites of cardiac involvement are mitral valve causing MVP and dilatation of the aortic root.

A preconception echo is done to determine the diameter of the aortic root. If it is > 40 to 45 mm, she has the risk of acute aortic dissection. Hormonal influence on connective tissue during pregnancy weaken the medial layer of the aorta and increase the possibility of aortic dissection.

Eisenmenger syndrome:

It is characterized by pulmonary hypertension secondary to right to left or bidirectional shunt through PDA, ASD, VSD. Increase in pulmonary artery pressure and decrease in PVR may cause right to left stunt and arterial blood oxygen desaturation. Mortality rate is 22 to 50 %. Hence pregnancy is contraindicated.

Cardiac arrythmias:

Significant maternal arrythmias are rare during pregnancy. One of the arrythmia is paroxysmal Supra Ventricular Tachycardia due to atrioventricular node reentry. Rate varies between 150 and 250 bpm. Women present with palpitations, shortness of breath, light headedness. ECG shows narrow QRS complexes. Treatment includes carotid sinus massage, Adenosine, Verapamil, Cardioversion.

Myocardial Conditions

Peripartum cardiomyopathy:

Diagnostic Criteria includes:

1. Heart failure in last month of pregnancy and five months postpartum
2. Absence of prior heart disease
3. No identifiable cause heart failure
4. Echo showing left ventricular ejection fraction < 45%, left ventricle end diastolic dimension > 2.7 cm/m^2, fractional shortening < 30%.

Women present with fatigue, palpitations, Orthopnea, paroxysmal nocturnal dyspnoea, cough. Physical examination shows tachycardia, cardiac arrythmias, pulmonary rales, peripheral edema. The exact cause is unknown.

Multiple etiologies have been proposed for PPCM including inflammation, viral myocarditis, abnormal immune or hemodynamic response to pregnancy, increased oxidative stress, malnutrition, cardiomyocyte-specific deletion of the transcription factor signal transducer and activator of transcriptions 3 (STAT3) protein and genetic factors.

It has a high mortality rate of 25 to 50 %. Bed rest, Beta blockers, ACE

inhibitors, diuretics, digitalis are recommended during pregnancy. Subsequent pregnancy carries a recurrent risk of 25 to 50 %.

Coronary Artery Disease:

Pregnancy may be considered in women with known CAD, if there is no residual ischaemia and left ventricular ejection fraction > 40 %. Statins should be stopped before conception and ACE inhibitors are avoided. During pregnancy most common cause of myocardial infarction is due to spontaneous coronary artery dissection.

High progesterone levels may lead to structural changes in the collagen of the vessel wall. Ergometrine may lead to coronary vasospasm and ischaemia. Diagnosis is made by ECG and elevated cardiac markers. CHF, angina, severe arrythmia may complicate the course of pregnancy.

References

1. Cunningham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, Casey BM, Sheffield JS (eds). William's Obsterics 25th edition; 2018; chapter 49 Cardiovascular Disorders.
2. Adams JQ, Alexander AM Jr. Alterations in cardiovascular physiology during labour. Obstet Gyneocl. 1958;12:542-9.
3. Lee W. Cardiorespiratory alterations during normal pregnancy. Crit Care Clin. 1991;7:763-75.
4. Cardiac disease in pregnancy; practical guide to high-risk pregnancy South Asian perspective, 4th edition, 2015.
5. Silversides CK, Colman JM, Sermer M, et al. Cardiac risk in pregnant women with rheumatic mitral stenosis. Am J Cardiol. 2003;91:1382-5.

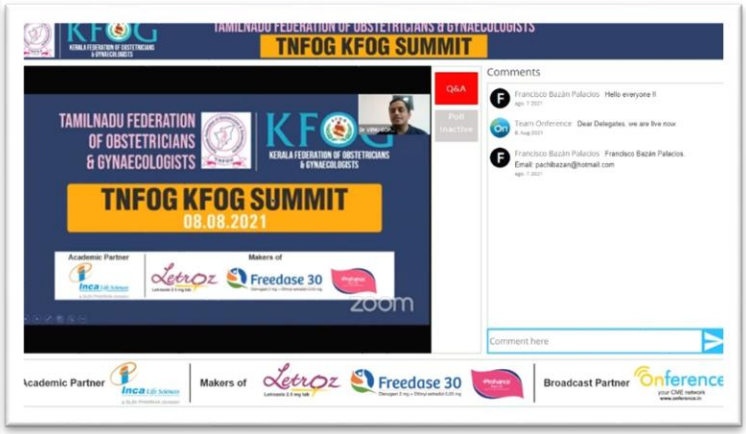
TNFOG

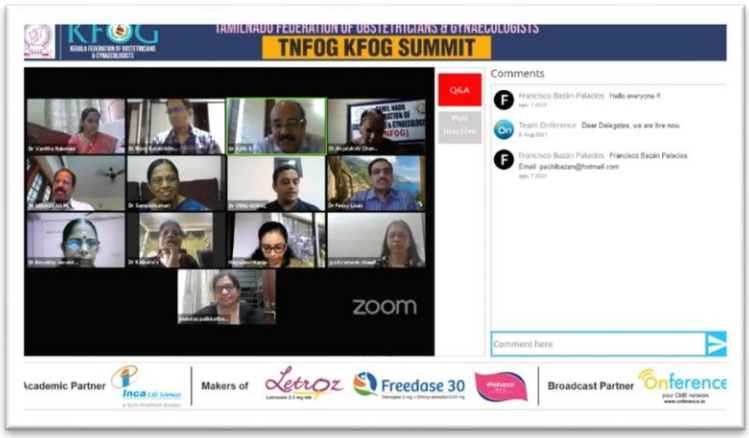
Past Events

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

TNFOG Infertility Committee

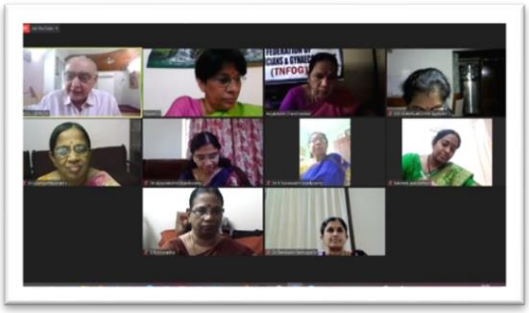
8th Aug 2021

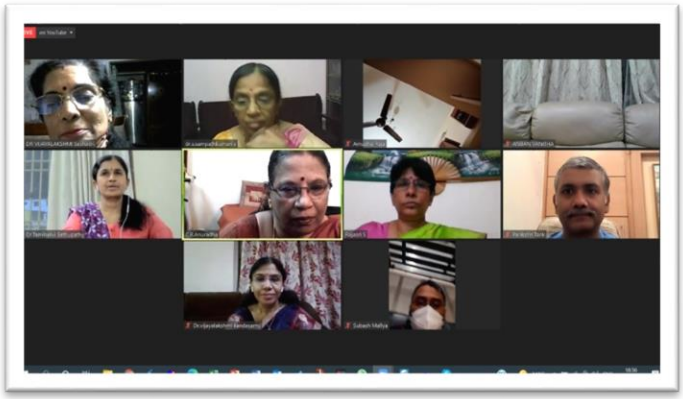




TNFOG Marathon CME on Fibroids

12th Aug 2021





QUIZ WINNERS

Session	Dr Name	City	Phone Number	Time
1	Dr. Nandhini Raman	Chennai	8939310629	17:19:39
2	Dr. Priyanga Soodimuthu	Thanjavur	9789282207	18:40:14
3	Dr. Jeyasudha Rathinam	Puducherry	9443871611	19:25:48

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

Paramedics Training CME

TNFOG with Tamilnadu Women Doctor's Wing

14th - 20th August 2021 | **4.30 - 6.00 PM**

Chief Guest

Dr. Hrishikesh D Pai
President Elect,
FOGSI (2022-23)

Special Guest

Dr. Hema Divakar
Vice Chair,
FIGO PNCD Committee

Guest of Honour

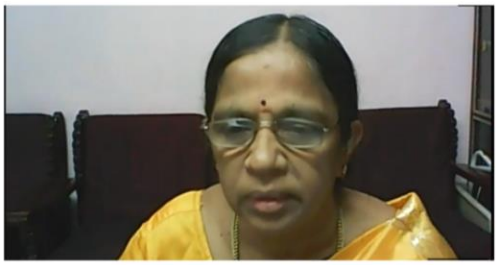
Dr. L. Yesodha
Chairman, National
IMA WOMEN DOCTOR's wing

Dr. Maragathamani
V.P North Zone,
TN IMA

Speakers

14th Aug'21	15th Aug'21	16th Aug'21	17th Aug'21	18th Aug'21	19th Aug'21	20th Aug'21
Topic : Normal labour	Topic : PIH	Topic : Anaemia in Pregnancy	Topic : PPH	Topic : Partogram	Topic : Neonatal resuscitation & Breast feeding	Topic : Puerperal sepsis & PPIUCD
						
Dr. Poorni Narayanan	Dr. S. Sampath Kumari	Dr. T. K. Shaanthy Gunasingh	Dr. Poorni Narayanan	Dr. V. Kasthuri	Dr. Meenakshi Subramaniam	Dr. Revathy Janakiram

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS



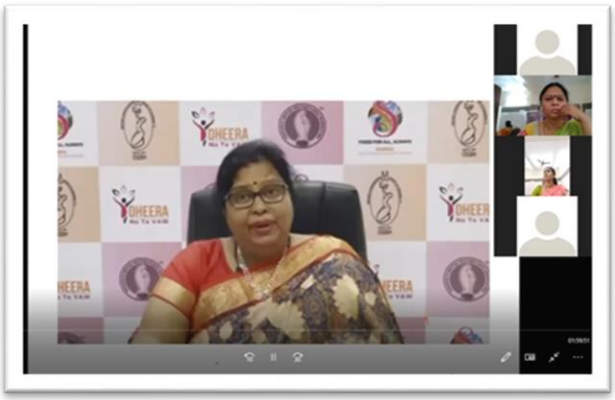
WINNERS

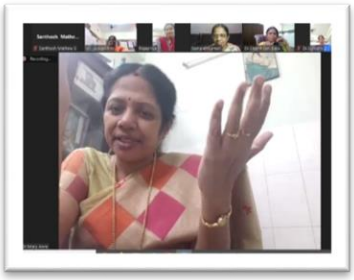
Name	City	Phone Number	Mail ID	Marks (70)
Arun Hospital	**madurai**	4545777	arunhospitalmdu9@gmail.com	66
Thenmozhi	**Sivakasi**	9344403414	thenmozhigg1990@gmail.com	61
Selvi	**Vellore**	9952331842	pselvi250@gmail.com	59

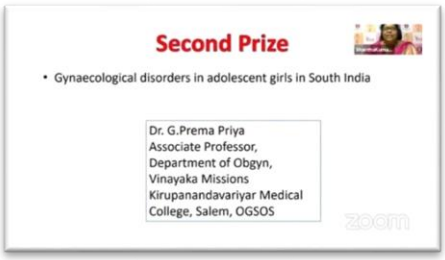
Adolescent Health Week

(Theni, Chennai, Dindigual, Viruthunagar)

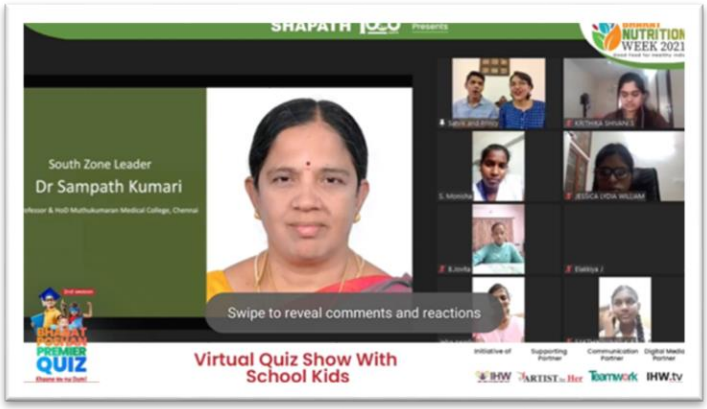
1st week of September

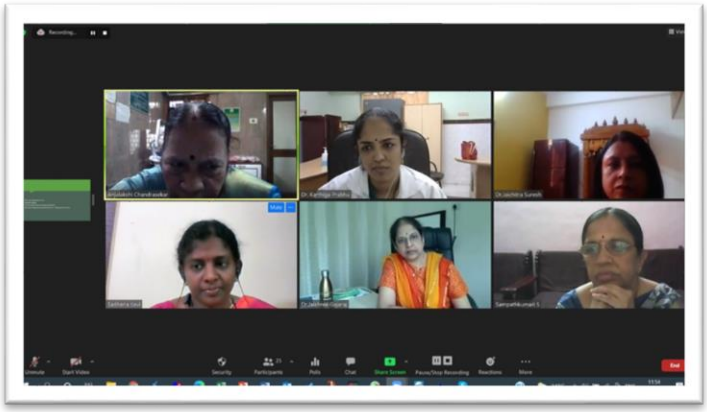






Awareness Programs





TNFOG
Upcoming Events

Magalir Nalam CME
on LSCS

18th September 2021

Time: 2:30 PM to 6:30 PM

TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGISTS

INNOVATIONS, SCIENCE WITH EXCELLENCE

Shield CONNECT

www.shieldconnect.in