Theme :
Medical Disorders in Pregnancy

Chief Editor
Dr. Sadhana Gupta

Guest Editor
Dr. Arun Arora

Jt. Editor
Dr. Amrita S Jaipuriar

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Chairman's MESSAGE

Dear Esteemed Colleagues,

It is with great pleasure that we present the third bulletin of the North India Gynaecologist Forum focusing on the crucial theme of "Medical Disorders in Pregnancy." As guardians of maternal and fetal health, it is imperative for us to delve into the complexities of managing medical conditions during pregnancy.

In this bulletin, we aim to provide a scholarly discourse on various medical disorders encountered in pregnancy, encompassing comprehensive diagnostic approaches, evidence-based management strategies, and multidisciplinary collaboration. From gestational diabetes to hypertensive disorders, from thyroid dysfunction to autoimmune diseases, each condition poses unique challenges requiring tailored interventions.

Through this scholarly endeavor, we endeavor to enhance our collective understanding, refine our clinical acumen, and ultimately improve pregnancy outcomes for the women under our care.

Let us continue to strive for excellence in maternal-fetal medicine, guided by science, compassion, and a relentless pursuit of optimal care.

Warm regards,

Dr. Sharda Jain
Founder & Chairperson NIGF

President & Chief Editor MESSAGE

It is moment of pride and pleasure to present 3rd issue of North India Gynae Forum (NIGF) bulletin to our members on the occasion of National Safe Motherhood Day 11 April, which is birth anniversary of Kasturba Gandhi.

Our country has travelled a long way on path of Safe Motherhood with gross decline of Maternal Mortality after our independence and particularly in last decade. Central & State Governments have shown political will in form of 108 Ambulance services, accessible obstetric care and many financial support schemes like Jannai Surakcha Yojna. Obstetrician & Gynecologist across the country deserve special appreciation & congratulations for making motherhood much safer in our country.

However Obstetric demography has radically changed in last decade. Women are entering pregnancy in late age, with medical comorbidities and on different medications. Many medical conditions are now medically & surgically treated well and these women can have joy of motherhood

It is unique challenge & it is need of time that every obstetrician & Gynecologist is well versed with basics of common medical disorders, how these medical disorders effect mother and fetus & how to optimize drug usage and obstetric decisions. Keeping this in mind we have kept theme of this issue on Medical Disorders in Pregnancy.

Our learned authors have presented updates on continuum of Obstetric care from preconception to postpartum period in common medical disorders like chronic kidney disease, heart disease, Epilepsy, jaundice which can be life threatening if improperly managed. Endocrine disorders like Thyroid Disorders, Gestational Diabetes Mellitus as well skin disorders need meticulous care throughout pregnancy. We hope and believe that it will benefit every Obs. & Gyn. and their patients. Our hearty thanks to all Authors, Guest editor Dr Arun Arora & Jt. editor Dr Amrita S Jaipuriyar.

With great pride we also present a worthy journey travelled together for various social, academic learning programs like Bimonthly Quiz, Monthly Kannon Ki Pathshala. We applaud our all-state chapters leaders who are taking NIGF at high levels in all dimensions.

Together we can achieve our mission of Unity, Excellence & Service.

Stay Tuned

Dr Sadhana Gupta

President NIGF & Chief Editor NIGF Bulletin

President Elect MESSAGE

3rd issue of NIGF bulletin is being Published I congratulate Dr Sadhna Gupta for her wonderful academic activities. The topic discussed is very important for all ob gyn practitioners.

The prevalence of medical problems in pregnancy is increasing because of a complex interplay between demographic and lifestyle factors, and developments in modern medicine. Women are delaying childbearing until later in life. Older women are more likely to have acquired medical disorders, such as hypertension and obesity; they are also at higher risk of gestational diabetes and venous thromboembolism. Medical and surgical advances have enabled women to become pregnant despite having chronic conditions that would previously have precluded pregnancy. Even in developed world Three quarters of maternal deaths are in women with co-existing medical complications. Pregnant women in their 40s are three times more likely to die than pregnant women in their early 20s. With ART technique older women 40+ is getting pregnant.

It is very necessary to understand medical disorder in pregnancy for safe ending of Happy mother and Healthy baby.

Happy Reading

Jai NIGF

Dr Ragini Agrawal
President Elect NIGF

Chronic Kidney Disease In Pregnancy

Dr. (Prof.) Mamta Dagar, Dr. Ifat Irshad
Institute of Obstetrics & Gynaecology
Sir Ganga Ram Hospital, New Delhi, India

Introduction:

Chronic kidney disease is often clinically and biochemically silent until renal impairment is advanced. The global prevalence of chronic kidney disease is approximately 13.4%, with a higher prevalence in women as compared to men[1]. The prevalence of chronic kidney disease in women of reproductive age is low, about 0.1–4%,[2] however the implications of pregnancy in a women with chronic kidney disease are many and could be severe. Chronic kidney disease represents a heterogeneous group of disorders characterized by alterations in the structure and function of the kidney. Its manifestations are largely dependent on the underlying cause and severity of the disease, but typically include decreased function, hypertension, and proteinuria, which can be severe. The various etiologies of the kidney diseases include glomerular diseases, vascular diseases, tubulointerstitial diseases and cystic diseases. Systemic diseases like diabetes, vasculitis, and systemic lupus erythematous often also involve the kidneys[3].

Pathophysiology:

Symptoms of chronic kidney disease are unusual until the glomerular filtration rate declines to < 25% of normal, and more than 50% of renal function is lost before serum creatinine rises above 120ìmol/l[3]. Women who become pregnant with serum creatinine values above 124 ìmol/l have an increased risk of renal dysfunction and poor pregnancy outcome. Chronic kidney disease is widely classified into five stages according to the level of renal function, table 1[4]. Chronic kidney disease in pregnancy have mostly classified women on the basis of serum creatinine values. Some women can be found to have chronic kidney disease for the first time during pregnancy. Around 20% of women who develop early pre-eclampsia (d"30 weeks' gestation), especially those with heavy proteinuria have previously unrecognised kidney disease.

Table 1: Stages of Chronic Kidney Disease[4].

Stage	Description	Estimated Glomerular Filtration Rate (GFR) (ml/min/1.73m)
1.	Kidney damage with normal or raised GFR	> _90
2.	Kidney damage with mildly low GFR	60-89
3.	Moderately low GFR	30-59
4.	Severely low GFR	15-29
5.	Kidney failure	<15 or dialysis

Renal anatomic and physiologic changes associated with pregnancy:

For maternal physiological adaptation during pregnancy the important haemodynamic, endocrinal and anatomic renal changes that occur during pregnancy are summarised as follows[5].

- Renal vasodilation and increased renal plasma flow (RPF) by 50%–80% above non-pregnant level.
- Renal hyperperfusion and hyperfiltration causing increased urine output, frequency, nocturia.
- Increased glomerular filtration rate (GFR) (by 25%–55%), causing increase in proteinuria and creatinine clearance with a decline in serum creatinine and blood urea nitrogen (BUN). The normal serum creatinine levels in pregnancy are usually between 0.4 and 0.9 mg/dL.
- Reduced tubular glucose reabsorption causing glycosuria (in 70% of nondiabetic mothers).
- Hypercalciuria.
- Increase in kidney length by 1-1.5cm[22], increased pelvicalyceal, ureteral, bladder and urethral morphological changes contributing to urinary stasis and vesicoureteral reflux (VUR).
- Symptoms mimicking cystitis.

- The kidneys increase the production of erythropoietin, active vitamin D, and renin in pregnancy.

A pictorial summary of the renal structural, physiological, and hormonal changes in pregnancy is demonstrated in Figure 1.[6]

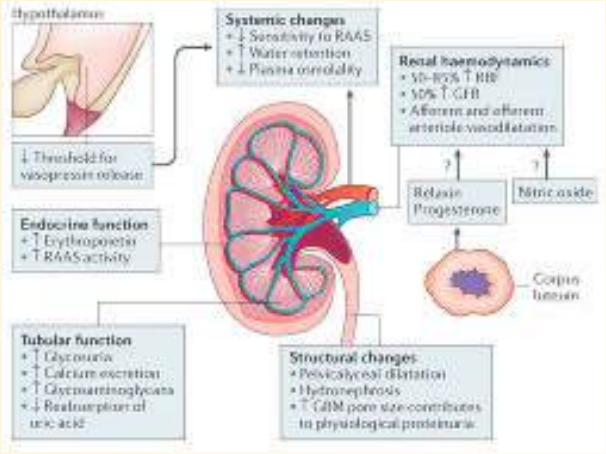

Fig 1: Physiological changes in the kidney during pregnancy. Reproduced from[6]

What is the effect of pregnancy on kidney disease?

Chronic kidney disease in pregnancy has the potential to hasten disease progression and lead to early progress to end-stage renal disease (ESRD). Various definitions of chronic kidney diseases have been defined, one of them is shown below in the table 2.[7]

Table 2. Chronic Kidney Disease Definitions: Chronic kidney diseases.

Degree of CKD	Serum Creatinine Level (mg/dL)	Creatinine Clearance (mL/min)	Stage of CKD
Mild	Less than 1.4	Greater than 70	1–2
Moderate	1.4–2.4	40–70	2–3
Severe	Greater than 2.4	Less than 40	3–4

Renal function declines as a result of pregnancy, and the degree of this decline depends on severity of the underlying renal disease. Women with well-preserved kidney function before pregnancy does not have a signicant renal function loss. Mild renal impairment with normal blood pressure, no/minimal proteinuria have lower risk for disease progression during pregnancy and long term[6]. However, women with advanced chronic kidney disease, should be counselled about the potential loss of kidney function during pregnancy and need for renal dialysis. Pregnancy termination does not reverse renal dysfunction. In women with severe renal insufficiency (creatinine greater than 2.4 mg/dL), significant pregnancy-related kidney dysfunction is observed, either during pregnancy or within 6 weeks postpartum, with 23% of women progressing to ESRD by 6 months postpartum[7]. Concomitant hypertension and proteinuria also contribute to the loss of renal function. When proteinuria exceeds 1 g/d in combination with compromised GFR (less than 40 mL/ min), there is accelerated loss of GFR[8]. ESRD is associated with increased mortality[7].

What is the effect of kidney disease on pregnancy?

Women with CKD appear to have at least a two-fold higher risk of developing adverse maternal outcomes as compared to women without CKD[9].The adverse maternal outcomes in women with CKD include higher rates of caesarean section, preterm delivery less than 37 weeks (89%), less than 34 weeks (44%), gestational hypertension, 10-fold increase in the risk of pre-eclampsia, eclampsia and maternal mortality[10]. Adverse fetal outcomes include (5-fold) premature births, IUGR, SGA, neonatal mortality, stillbirths, need for NICU admission and low birth weight.

Optimization strategies:

In women with advanced CKD, multidisciplinary care is important and should include senior obstetrician, nephrologists, maternal-fetal medicine specialists, neonatologists, and specialized neonatal intensive care units (NICU). For better outcomes, the care should begin in preconception period through delivery and then extending into the postpartum period. These principles of management extend to all stages of chronic kidney disease, including those women on dialysis and post-transplantation. It is best to diagnose the type of renal disease and optimize the management before pregnancy, which include; preconception counselling; assessment of renal function in pregnancy; management of hypertension with safe pregnancy options; Preeclampsia prophylaxis; treatment of proteinuria and the nephrotic syndrome; stabilization of any progression of kidney dysfunction,

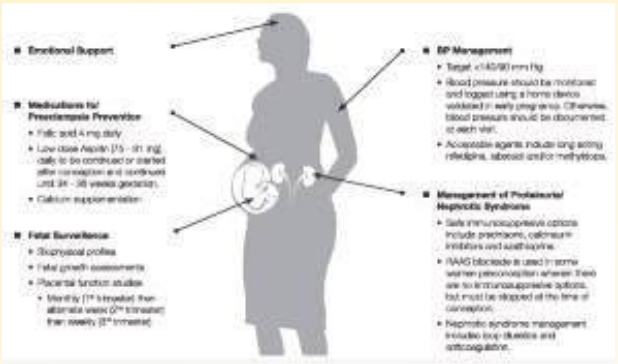

Fig 2: Antenatal care in women with chronic kidney disease[10]

Preconception Counselling[11]:

Pre-pregnancy counselling should be done by a multidisciplinary team which includes a consultant obstetrician and a nephrologist.

Women with CKD should be counselled about the increased risk of complications in pregnancy like more chances of caesarean delivery, pre-eclampsia, preterm birth, fetal growth restriction, and neonatal unit (NNU) admission.

Women with known inheritable renal diseases should be offered genetic counselling including inheritance risk, prognosis, and intervention options like pre-implantation genetic diagnosis.

The counselling for the optimisation of maternal and neonatal outcomes in women with CKD, should include[11]:

- Stabilising the disease activity in pregnancy on minimised doses of pregnancy safe medications.
- Optimising blood pressure control (< 140/90 mmHg) on pregnancy-safe medications
- Optimising glycaemic control in women with diabetes mellitus
- Minimise the risk of exposure to teratogenic medications like angiotensin converting enzyme inhibitors or angiotensin receptor antagonists (antihypertensives). Women should discontinue these medications owing to second- and third-trimester teratogenic risks, such as renal dysgenesis, perinatal renal failure, oligohydramnios, pulmonary hypoplasia, and hypocalvaria.[12] and switch to pregnancy-safe medications.
- Women with CKD stages 4 and 5 contemplating pregnancy should be offered pre-dialysis education.
- Immunosuppressive agents should be avoided in pregnancy and breastfeeding and include mycophenolate mofetil and cyclophosphamide and to be replaced with pregnancy-safe alternative agents.
- Making a treatment plan in the event of hyperemesis or disease exacerbation/relapse during pregnancy

Assessment of renal function in pregnancy[11]:

- Renal function in pregnancy is assessed using serum creatinine concentrations, as estimated GFR (eGFR) is not valid for use in pregnancy.
- Women with CKD should have formal quantification of proteinuria in pregnancy.
- Quantification of proteinuria is undertaken by protein: creatinine ratio (uPCR) or albumin: creatinine ratio (uACR).
- Twenty-four hour urine collection for quantification of protein is not required.

Management of hypertension with safe pregnancy options:

- The presence of hypertension increases the risk of adverse pregnancy outcomes like preeclampsia, preterm delivery, and fetal growth restriction[13].
- Blood pressure control ideally should be optimized before pregnancy.
- Medications compatible with pregnancy include methyldopa, labetalol, nifedipine, and hydralazine. Teratogenic agents should be discontinued.
- A blood pressure target of less than 135/85 mm Hg has been recommended for women with chronic kidney disease during pregnancy[11].

Preeclampsia Prophylaxis:

- Women with chronic kidney disease are at high risk for the development of preeclampsia, so low dose aspirin (75-150 mg) should be initiated before 16 weeks of gestation, and, with findings from the recent ASPRE (Combined Multimarker Screening and Randomized Patient Treatment with Aspirin for Evidence-Based Preeclampsia Prevention) trial, supports the suggestion [7,14].
- The World Health Organization recommends daily supplementation with 1.5–2 g of oral elemental calcium in populations with low dietary intake for the prevention of preeclampsia, but vitamin D supplementation requires further study[15].

Treatment of proteinuria and the nephrotic syndrome:

- Proteinuria should be minimized where possible before conception, and the best approach is to determine the underlying etiology of the renal disease before conception.
- Kidney biopsy is feasible in pregnancy but rarely

necessary. If the woman has received adequate preconception counseling, which typically would include a biopsy for definitive diagnosis. Biopsy can therefore be done only in early gestation, and indications include new-onset nephrotic syndrome, significant glomerular disease wherein confirmation of diagnosis will affect treatment choice, or a sudden deterioration in renal function. Beyond 30 weeks of gestation, however, the risks of kidney biopsy may supersede its benefits owing to technical challenges associated with a gravid uterus and the potential for coexistent preeclampsia.

- Once a diagnosis is ascertained, options for management include Immunosuppressive pregnancy-safe medicines, for diseases (lupus nephritis, vasculitis, membranous nephropathy, minimal change disease).
- Options for safe immunosuppression during pregnancy and breastfeeding include prednisone, azathioprine, and the calcineurin inhibitors[16].
- Stress doses of short-acting glucocorticoids during labor and delivery are necessary in those maintained on long-term corticosteroids to manage suppression of the hypothalamic-pituitary-adrenal axis.
- Renin-angiotensin-aldosterone system blockers discontinued before 8 weeks of gestation for non immunological diseases, like diabetic nephropathy, reflux nephropathy, hypertensive nephrosclerosis. Captopril and enalapril, have been determined to be negligible in breast milk, so renin-angiotensin-aldosterone system blockers can be reinstituted in the early postpartum period to manage proteinuria[17].
- For significant flares of renal disease during pregnancy, pulse steroids followed by combination immunosuppression is a common approach.
- Women with lupus or vasculitis should be advised to wait until their disease is quiescent for at least 6 months before conceiving. They should be advised to take hydroxychloroquine in pregnancy unless it is contraindicated and monitoring for disease activity during pregnancy is necessary[11].
- Women with severe proteinuria causing nephrotic syndrome ie proteinuria greater than 3 g with low albumin, hypercoagulability and incapacitating peripheral edema may be seen, which can be treated with conservative therapy which include, use of compression stockings and elevation of the extremities, careful use of loop diuretics like furosemide. Albumin infusions in extreme cases may be necessary[18] .
- Pregnancy is a prothrombotic state[19], and in patients with severe hypoalbuminemia there is a significantly increased risk of venous thromboembolism1[20] .
- Women with nephrotic-range proteinuria (uPCR > 300 mg/mmol or ACR > 250 mg/mmol) be offered thromboprophylaxis with low molecular weight heparin in pregnancy and continued for at least 6 weeks postpartum period unless there is a specific contraindication including risk of labour or active bleeding.[11].

Stabilization of any progression of kidney dysfunction:

Strategies in chronic kidney disease include avoidance of nephrotoxic agents; optimizing dosing of commonly used medications; management of anemia; bone mineral metabolism; and management of acid–base and electrolyte imbalances.

- Nephrotoxic medications among tocolytics such as indomethacin or antimicrobial agents such as gentamicin should be avoided, nonsteroidal antiinflammatory medications for postpartum pain control also should be avoided. Magnesium, a frequently used medication for eclampsia prevention or fetal neuroprotection, is excreted by the kidneys, and, magnesium toxicity presents a significant risk in women with advanced chronic kidney disease, especially those on dialysis. Thus, regular assessment of serum magnesium concentrations and deep tendon reflexes and a lower constant infusion rate (eg, 1 g/h) are often indicated.
- Anemia is common in pregnant women with CKD, so oral iron, parenteral iron or both can be given, erythropoietin stimulating agents are also recommended[11].
- Hypocalcemia and hyperphosphatemia due to secondary hyperparathyroidism from advanced chronic kidney disease can be treated safely with oral calcium carbonate and vitamin D analogues (5).
- Women with chronic kidney disease may have electrolyte abnormalities, a low-potassium diet should be initiated followed by binding resins can be used if needed. This may be an indication to initiate dialysis.
- There are increased chances of maternal acidosis and maternal serum pH should be maintained at greater than 7.2. Sodium bicarbonate therapy to be given and is an indication for dialysis in extreme cases.
- Volume depletion or volume overload should be highlighted and fluid balance is managed with the aim of maintaining normal fluid volume, avoiding dehydration and pulmonary oedema.

Peripartum care[11]

- Owing to increased risk of adverse pregnancy outcomes, more frequent prenatal assessments may be necessary to allow close maternal and fetal monitoring, with additional specialist input.
- Women with CKD have observations taken and documented during any hospital admissions. These include temperature, heart rate, blood pressure, respiratory rate, and oxygen saturation. An early warning score should be calculated and actioned appropriately.
- The timing of birth for women with CKD is determined by obstetric indications, with consideration of renal factors including deteriorating renal function, symptomatic hypoalbuminaemia, pulmonary oedema, and refractory hypertension.
- A rapid and severe decline in renal function is an indication for preterm delivery or termination of pregnancy.
- In the absence of maternal or fetal compromise, consideration should be given to delivery at or near term, with cesarean delivery reserved for usual obstetric indications.

Postnatal care

- Women with CKD should not be given non-steroidal anti-inflammatory medicines.
- They should have a planned early postpartum renal review.
- They should be prescribed medications that are compatible with breastfeeding whenever possible.
- These women should offered safe and effective contraception postpartum and receive updated pre-pregnancy counselling before future pregnancies[11].

Pregnancy and end-stage renal disease

In the population of women with ESRD on dialysis, conception and maintenance of pregnancy were historically infrequent and complex events. Fertility rates are low in those on hemodialysis and even lower in women on peritoneal dialysis[21]. The reason was thought to be impaired pituitary release of luteinizing hormone contributing to anovulation. However, hemodialysis in the context of pregnancy is becoming more common, it is influenced by the use of more biocompatible membranes and the increasing use of erythropoietin-stimulating agents. More intensified dialysis prescriptions, can be considered a reproductive choice for some women when transplantation is not imminent.

Despite improvement in live birth rates, pregnancy with intensified dialysis is associated with many risks such as Preterm birth, preeclampsia, low birth weight, and fetal growth restriction, necessitating careful care and follow-up by an interdisciplinary care team. Details of the dialysis management for these women is beyond the scope of this article.

Conclusion:

Management of pregnancy in chronic kidney disease represents a complex situation with increased risks of adverse maternal and perinatal outcomes. Early recognition of early-stage chronic kidney disease is important. Commencing the mangement in pre-pregnancy period, even in women with advanced chronic kidney disease and those on dialysis or with a kidney transplant are benefited from combined care with multidisciplanary team which include nephrology specialists, high-risk obstetricians or maternal-fetal medicine specialists. Access to neonatal intensive care unit is also critical. Finally, expert knowledge and skill provided by a multidisciplinary team of health care professionals is beneficial for optimizing pregnancy outcomes.

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"God could not be everywhere, and therefore he made mothers."

–Rudyard Kipling

Skin Disorders In Pregnancy

Dr. Geetu Mahajan
Associate Professor
JIET Medical College & Hospital
Author

Dr. Arunav Arora
Resident
GMC, Jammu
Co-author

Dr. Arihant Arora
Resident
GMC, Jammu
Co Author

Distinct heterogenous group of cutaneous disorders that appear during pregnancy and impact the health of the pregnant woman and potentially the fetus are called dermatoses of pregnancy. These disorders occur as a result of interaction of multiple factors which include an increase in hormone levels, intravascular volume expansion, and skin compression from the pregnant uterus. These skin changes can be classified into three groups:

1. Physiological changes
2. Pregnancy Specific Dermatosis
3. Pre-existing skin disease

Physiological Changes	Pregnancy specific Dermatosis
Pigmentary Changes	Atopic Eruptions of pregnancy
Hair and Nail Changes	Polymorphic Eruptions of Pregnancy
Vascular Changes	Pemphigoid gestationis
Glandular Changes	Intrahepatic Cholestasis of Pregnancy

PHYSIOLOGICAL CHANGES (Table1): These are caused by hormonal changes during pregnancy and include:

Physiological skin changes during pregnancy (Table 1)

Pigmentary	Nail Changes	Hair Changes	Vascular Changes	Glandular changes
Hyperpigmentation	Britleness	Thickening of scalp hair	Telangectasias	Hyperhidrosis
Linea Nigra	Increased Growth	Hirsutism	Spider Naevi	Miliaria
Melasma	Subungal Keratosis	Androgenic Alopecia	Varicose Veins	Montgomery Tubercles
Stria Gravidorum	Onycholysis		Non Pitting Edema	
	Transverse Grooves		Palmer Erythema	

PIGMENTARY CHANGES

Hyperpigmentation: This is most common presentation of pregnancy caused due to increased levels of MSH, estrogen or progesterone. Nearly all women experience some degree of hyperpigmentation during pregnancy. These changes are more pronounced in women with a darker complexion. The areolae, axillae and genitals are most commonly affected. Scars and naevi may also darken.

Linea Nigra: The line that often forms when the abdominal linea alba darkens during pregnancy. This is often accompanied by displacement of umbilicus to the right, known as the "ligamentum teres sign"

Linea Nigra

Melasma (chloasma or mask of pregnancy) may be the most cosmetically troublesome skin condition associated with pregnancy. The condition occurs in up to 70 percent of pregnant women[2]. Exposure to sunlight and other ultra violet radiation worsens melasma. Although no specific treatments are indicated during pregnancy, physicians can

reassure patients that melasma resolves postpartum in most cases.

Melasma

Striae Gravidorum develops in up to 90% of women during sixth and seventh month of pregnancy.[2] These are seen as reddish or bluish depressed streaks, usually on abdomen but also on the breast and thighs.

Striae Gravidorum

HAIR CHANGES: An increase in growth and production of hair is common during pregnancy.

Hirsutism: Many women experience some degree of hirsutism on the face, limbs, and back caused by endocrine changes during pregnancy which generally resolves postpartum, although cosmetic removal may be considered if the condition persists.

Thickening of scalp hair: Pregnant women also may notice mild thickening of scalp hair. This is caused by a prolonged active (anagen) phase of hair growth. Postpartum, scalp hair enters a prolonged resting (telogen) phase of hair growth, causing increased shedding (telogen effluvium), which may last for several months or more than one year after pregnancy.[2,3]

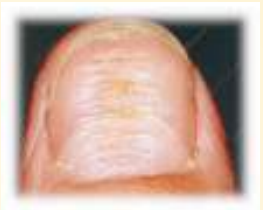
Androgenic Alopecia: A few women with a tendency toward androgenic alopecia may notice fronto-parietal hair loss, which may not resolve after pregnancy.

Hirsuitism

Androgenic Alopecia

NAIL CHANGES: Nails usually grow faster during pregnancy. Pregnant women may experience increased brittleness, transverse grooves, onycholysis, and subungual keratosis.[2,3] Most of these conditions resolve postpartum, although physicians can reassure patients and promote good nail care.

Transverse Grooves



Subungal Keratosis

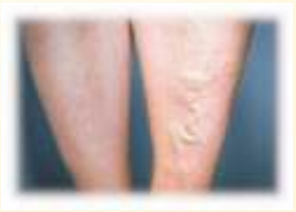
VASCULAR CHANGES: Vascular changes are caused by increase in vascularity associated with high estrogen levels and increased blood volume.

Spider Telangectasias: They are common in areas drained by superior venacava which include face, neck, upper chest and arms.

Palmer erythema is common, affecting 70% of light complexion women with and 30% of women with darker skin types.

Palmer Erythema

Varicose Veins of the legs, hemorrhoids, and vulvar varicosities are frequent complications of pregnancy.



Varicose Veins

Non pitting edema of legs, eyelids and extremities due to increased hydrostatic pressure can occur in up to 50% of normal pregnancies. Increased blood flow and instability of pelvic vessels may cause vaginal erythema (Chadwick's sign) and a bluish discoloration of the cervix (Goodell's sign).[2] Vasomotor instability also may cause facial flushing; dermatographism; hot and cold sensations;

and marble skin, a condition characterized by bluish skin discoloration from an exaggerated response to cold.[3]

GLANDULAR CHANGES

Hyperhidrosis and Miliaria: Eccrine sweat gland activity increases across the body except on the palms. Sebaceous gland excretion of sebum increases due to increased level of progesterone and androgens. Montgomery glands that provide lubrication to the nipples and areolae enlarge during pregnancy and appear as papules on the areolae (Montgomery tubercles).

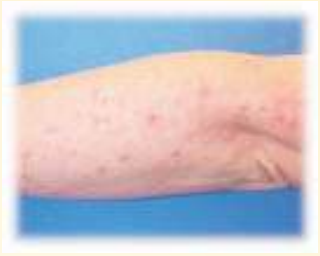
PREGNANCY SPECIFIC DERMATOSIS[3] include heterogenous group of pruritic skin eruptions which are seen only in pregnancy. Most of these are benign and resolve spontaneously in the postpartal period but a few of these are associated with fetal complications.[4] The most recent classification was proposed by Ambros-Rudolph et al[5] in 2006. They include:

- Atopic Dermatitis of pregnancy
- Polymorphic Eruption of pregnancy
- Intrahepatic Cholestasis of pregnancy
- Pustular Psoriasis of pregnancy
- Pemphigoid Gestationis

Atopic eruption of pregnancy (AEP) is the most common of these disorders, occurring 1 in 300 pregnancies[1]. It includes three conditions-Eczema, Prurigo and Pruritic folliculitis of pregnancy.

Eczema in pregnancy: There is high incidence of eczema in pregnancy for first time[6]. The reason for increased incidence was cited to be due to immunological changes in pregnancy. T-helper 2 (Th2) shift associated with pregnancy may favor the exacerbation of atopic dermatitis. Eighty percent of the affected patients experience first episode of atopic eczema during pregnancy. These patients have atopic background with raised total serum IgE levels. The skin lesions start during first and second trimester and affect all parts of the body including face, palms and soles.

Prurigo of Pregnancy (PP): This occurs one in 300 pregnancies and is characterized by pruritic, excoriated papules and nodules on the extensor surfaces of the legs and upper arms. The time of onset is variable and can occur in all trimesters. There may be underlying history of atopy. No adverse effects are seen in the mother and fetus.

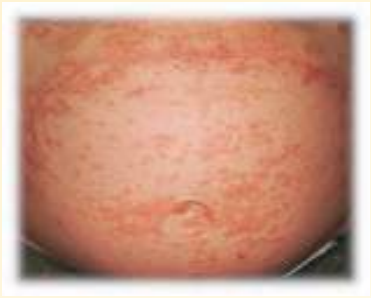


Prurigo of pregnancy

Pruritic Folliculitis (PF): It affects 1 in 3000 pregnancies and occurs in the second and third trimester of pregnancy. It is characterized by acnieform eruptions consisting of multiple, pruritic, follicular papules and pustules typically on the shoulders, upper back, arms, chest, and abdomen. The skin lesions resolve in 1-2 months following delivery. It is not associated with any maternal or fetal morbidity. The diagnosis is made by excluding other common rashes. Atopic eruptions are benign skin conditions and do not carry any fetal risk. They usually resolve in early postpartal period. AEP respond well to emollients and moderately potent steroids and oral histamines. Pruritic folliculitis responds well to topical benzoyl peroxide and has been treated with narrowband UV B phototherapy.

Pruritic Folliculitis

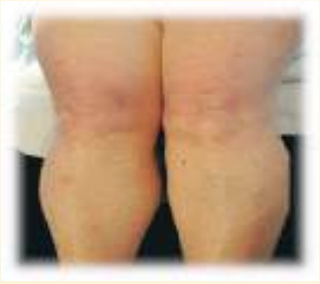
Pruritic urticarial papules and plaques of pregnancy (PUPPP): Incidence is 1 in 120 to 1 in 240[7] and is the second most common skin dermatosis in pregnancy after atopic eczema. This is characterised by itchy, erythmatous papules that coalesce into plaques that are classically found on the abdomen, sparing the umbilical area, and are found primarily in the abdominal striae. It may spread to the breasts, upper thighs and arms. The face, palms, soles and mucosal surfaces are usually spared. Onset is in late third trimester and resolves with delivery and rarely recurs in subsequent pregnancy. Mother and fetus are not affected.[8,9] The etiology of PUPPP is still unknown. Placental products, hormonal alterations and damage to connective tissue with subsequent conversion of nonantigenic molecules to antigenic ones, have been proposed as possible causes. Systemic steroids may be used for severe cases of PUPPP.[8,9]



PUPPP

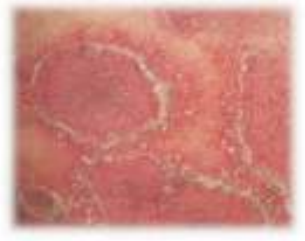
Intrahepatic Cholestasis of Pregnancy (ICP) (Obstetric cholestasis, Pruritus Gravidorum)

ICP was first described by kehr in 1907.[6] Incidence is 10-150 cases per 10,000 pregnancies.[10] Patients complain of sudden onset of pruritus beginning from the palms and soles and later generalizing to the whole body. Skin lesions are secondary to itching and range from excoriations to prurigo nodularis, extensors are more severely involved. Jaundice is seen in 20% cases only.[11]Clay colored stools, dark urine and hemorrhage secondary to vitamin K malabsorption can occur. Family history can be elicited in half of the cases and an association with multiple gestation is described. Resolution of ICP occurs soon after delivery. Recurrence in subsequent pregnancies is seen in 45 -70% cases. Diagnosis is made by increased serum bile acid levels, transaminases are also elevated.Prothrombin time may be prolonged. It is associated with fetal morbidity including premature births in 20-60% cases, intrapartal fetal distress including meconium aspiration in 20-30% and fetal mortality in 1-2%. Risk is more if serum bile acid levels exceed 40 micromoles per litre. Ursodeoxycholic acid , given in dose of 15mg/kg orally daily is the only proven therapeutic agent that decreases fetal mortality.[12]



ICP

Pemphigoid Gestationis (Herpetic Gestationis)(HG): Dermatitis herpetiformis of pregnancy was first described in 1811 and was named herpes gestationis by Milton in 1872. HG is a rare, self limited autoimmune bullous disorder that presents mainly in late pregnancy or the immediate postpartum period but can appear in any of the three trimesters. The incidence of herpes gestationis is 1 in 50,000 pregnancies. The condition is linked to the presence of HLA-DR3 and HLA-DR4. It has been suggested that the disease could be triggered by a placental antigen that causes cross reaction with skin antigens. This explains the onset of disease in the peri-umbilical region. Clinically HG presents with intense pruritic erythematous urticarial papules and plaques that develop typically on the abdomen. Sites such as face, mucous membranes, palms, and soles are spared. In the "prebullous" stage, differentiation between HG and polymorphic eruption of pregnancy is somewhat difficult but investigation solves the problem. It occurs in second or third trimester and improves during the later phase of pregnancy but sometime it may flare at the time of delivery or postpartum. Complete resolution is seen in weeks to months after postpartum. The antenatal risks are small for date babies and premature birth. 5% to 10% of babies will develop lesions from passive transplacental transmission of antibodies. Pemphigoid gestationis is rarely associated with hydatidiform moles and choriocarcinoma.[13] Mild pruritis can be treated with oral antihistamines but patients with severe disease may require systemic steroids. High dose immunoglobulins are also used.



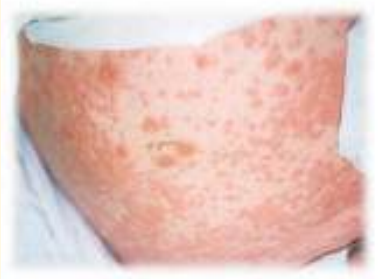
Pemphigoid Gestationis

Pustular psoriasis of pregnancy (PPP) is a life threatening condition for both the pregnant mother and fetus, and must be efficiently and accurately diagnosed and treated. Controversy exists about PPPs inclusion as a fifth dermatosis of pregnancy as it is more likely to be a variant of disease rather than a pregnancy specific eruption. However, this has been included in dermatosis of pregnancy owing to the importance of early recognition and treatment.[14]

The clinical presentation of PPP includes the formation of sterile cutaneous pustules studded on erythematous patches in the skin folds. Within one day, the pustules coalesce to form large dry plaques, which desquate in large sheets episodically. The disease tends to spread

centrifugally to the extremities, while sparing the face, palms and soles.[15] This is accompanied by systemic changes including fever, neutrophilia, electrolyte abnormalities and elevated inflammatory markers like CRP, WBC count. PPP tends to occur in the early part of the third trimester of pregnancy and resolves after parturition. The likelihood of recurrence in subsequent pregnancies is significant.

PPP can have significant effects on fetal health, leading to IUGR and even miscarriage and stillbirth. When a woman has systemic symptoms the fetus should be closely monitored due to increased risk of fetal anomalies and fetal demise.



Pustular Psoriasis

Pre-existing Skin Conditions like atopic dermatitis, psoriasis, candidial and other fungal infections, cutaneous tumors including molluscum fibrosum gravidorum and malignant melanoma may change during pregnancy. Atopic dermatitis may be related to prurigo of pregnancy and usually worsen, but may improve during pregnancy. Psoriasis is more likely to improve than worsen. Fungal infections generally require longer duration of treatment during pregnancy[16]. Soft tissue fibromas can occur on face, neck, upper chest and beneath breast during late pregnancy. These generally disappear post-partum. The effects of pregnancy on the development of malignant melanoma is debatable[17].

Key Points

- Multiple cutaneous disorders appear during pregnancy as a result of interaction of multiple factors.
- Most common among them are physiological changes and pruritus and they have to be differentiated from the pathological conditions that are specific to pregnancy.
- Common physiological conditions include pigmentation, vascular, nail and hair changes. No treatment is required for them as they subside during postpartum period.
- Pruritus in pregnancy should never be neglected and should always lead to a precise work up of the patient, which may be the symptom of the specific dermatoses of pregnancy. Careful history taking and examination will help to identify each condition clinically.
- The physical examination should focus on the distribution and morphology of the lesions. Involvement of striae is seen commonly in pruritic urticarial papules and plaques of pregnancy (PUPPP) but not in other dermatoses. Nodular lesions on the limbs are commonly seen in PP, whereas a follicular distribution is characteristic for PF or acne. Urticarial lesions suggest PUPPP or HG, whereas vesicular lesions can be seen in HG, herpes simplex/zoster, eczema, and occasionally in PUPPP.
- Most skin conditions resolve postpartum and only require symptomatic treatment. Liberal application of topical emollients and antihistamines should be advised in pregnancy associated pruritus. However, there are specific treatments for some conditions. Antepartum surveillance is recommended for patients with intrahepatic cholestasis of pregnancy, impetigo herpetiformis, and pemphigoid gestationis

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"Mother is the heartbeat in the home;
and without her, there seems to be
no heartthrob.

–Leroy Brownlow

"A mother is she
who can take the place of all others
but whose place no one else can take.

–Gaspard Mermillod

Valvular heart diseases in pregnancy



Dr. Natasha Gupta
Assistant Professor
All India Institute of Medical Sciences, Vijaypur, Jammu

Dr. Akshita Gupta
Senior Resident
All India Institute of Medical Sciences, Vijaypur, Jammu

Dr. Gurpreet Kour
Senior Resident
All India Institute of Medical Sciences, Vijaypur, Jammu

Introduction

Approximately 2% of pregnancies are complicated by cardiac diseases, with one-fifth of maternal deaths in developing nations attributed to these conditions. Valvular heart disease (VHD) accounts for one-third of cardiac cases during pregnancy in developing countries, while congenital heart disease is the dominating cause of heart disease in pregnancy in the western world [1]. In India, RHD contributes around 69 percent of cardiac diseases in pregnancy [2]. VHD can be acquired or congenital, both posing special challenges in the management. The degree and severity of VHD determines the risk of maternal and fetal complications. As a rule, regurgitation lesions are better tolerated than stenotic lesions and right-sided (pulmonary/tricuspid valve) lesions are better tolerated than left-sided (mitral/aortic) lesions. Preconception counselling also is an important domain for these patients. Managing valvular heart disease during pregnancy requires a collaborative approach, involving cardiologists, anaesthesiologists, and maternal-fetal medicine specialists to address the unique challenges and ensure optimal care for both the mother and the baby.[3]

Hemodynamics in pregnancy

During pregnancy, significant changes occur in the body's hemodynamics which can result in decompensation in cases of severe valvular disease **(Table 1)**. Each uterine contraction transfers 300-500 ml of blood to the maternal circulatory system through auto-transfusion during labor [4]. Labor and delivery are associated with sudden hemodynamic changes and an increase in oxygen consumption. The maternal body also undergo major alterations within the first 24 hours following childbirth **(Table 2)**. The uterus no longer compresses the inferior vena cava, leading to an increase in preload. These changes predispose women with underlying VHD to increased risk of heart failure. Furthermore, there is an increased risk of thromboembolism during pregnancy and postpartum period up to 12 weeks.[5]

Table 1. Physiological changes during pregnancy

Parameters	Variation	Comments
Cardiac output	Increase	40-50%
Blood volume	Increase	30-40%
Oxygen consumption	Increase	100 %
Heart rate	Increase	
Blood pressure pressure	Decrease	Diastolic blood > Systolic blood pressure
Systemic vascular resistance	Decrease	20-22%

Table 2. Physiological changes in the immediate postpartum period

Parameters	Variation	Comments
Cardiac output	Increase	Immediate increase (60-80%) followed by rapid decrease, returns to normal levels after few weeks.
Blood volume	Decrease	Due to blood loss
Heart rate	Decrease	Return to pre-pregnant level by 6 weeks postpartum
Stroke volume	Increase	Remain high up to 24 hours post partum
Systemic vascular resistance	Increase	Returns to pre-pregnant state in 2 weeks
Blood pressure	Decrease	Diastolic Blood Pressure > Systolic Blood Pressure

Preconceptional counselling

It's crucial to engage both a cardiologist and a maternal-fetal specialist before planning pregnancy.

A careful and elaborated history and examination including tests for connective tissue disorders should be performed. The baseline cardiac function is to be assessed by 12-lead electrocardiogram and echocardiogram. Exercise stress testing can be an effective tool for assessing exercise capacity and risk of development of arrhythmias. Cardiac MRI or computed tomography is indicated to evaluate valve function, structural anatomy, and any concomitant aortopathies, not visible in an echocardiogram.[6] Risk stratification of women with VHD is best done in the pre-conceptional period.

Prior to conception, medications with teratogenic effects, such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and statins, should be adjusted or substituted as necessary. Beta-blockers are usually considered safe in pregnancy. In first trimester change of anti-coagulation therapy may be considered for patients with mechanical heart valves on warfarin.

Risk assessment

Three widely recognized models which can help in assessing the risk of cardiac complications during pregnancy are CARPREG risk score (CARdiac Disease in PREnancy), ZAHARA risk score (for pregnant women with congenital heart disease) and modified WHO risk assessment score **(Table 3, Table 4, Table 5)**. The universally used functional classification is New York Heart Association (NYHA) Classification.

Table 3: CARPREG Risk Score II (Cardiac disease in pregnancy risk assessment)

Risk factor	Points
Prior cardiac events or arrhythmias	3
Poor functional class (NYHA class III/ IV) or cyanosis	3
Mechanical heart valve	3
Ventricular dysfunction (Ejection fraction < 55%)	2
High risk left-sided valve disease /left ventricular outflow tract obstruction	2
Pulmonary hypertension	2
Coronary artery disease	2
High risk aortopathy (ascending aorta diameter >45 mm)	2
No prior cardiac intervention	1
Late pregnancy assessment	1

CARPREG Score	Risk of cardiac complications during pregnancy
0 to 1	5 %
2	10%
3	15%
4	22%
>4	41%

Table 4: Zahara Prediction score

Predictors	Points
Prior arrhythmias	1.5
NYHA class >II	0.75
Left heart obstruction (Peak gradient >50 mmHg)	2.5
Cardiac medication before pregnancy	1.5
Systemic AV valve regurgitation	0.75
Pulmonary AV valve regurgitation	0.75
Mechanical valve prosthesis	4.5
Cyanotic heart disease (corrected / uncorrected)	1.0

Abbreviations : AV = atrioventricular; NYHA = New York Heart Association

ZAHARA SCORE	Risk of cardiac complications during pregnancy
0	2.9 %
0.5 to 1.5	7.5%
1.5 to 2.5	17.5%
2.5 to 3.5	43.1%
>3.5	70%

Table 5: Modified World Health Organization (WHO) pregnancy risk classification

Class	Condition	Risk
I	• Uncomplicated, mild pulmonary stenosis/patent ductus arteriosus /mitral valve prolapse • Successfully repaired simple lesions (ASD or VSD, PDA, anomalous pulmonary venous drainage) • Atrial or ventricular ectopic beats	No detectable increase risk of maternal mortality & no/mild increase risk in morbidity
II	• Unrepaired ASD or VSD • Repaired tetralogy of Fallot • Most arrhythmias	Small increased risk of maternal mortality & moderate increase risk in morbidity
II–III	• Mild left ventricular impairment • Hypertrophic cardiomyopathy • Bioprosthetic valve • Native VHD not considered WHO I or IV • Marfan syndrome where dilatation of ascending aorta < 40 mm • Bicuspid aortic valve where ascending aorta diameter < 45 mm • Repaired coarctation of aorta	Borderline between II & III)
III	• Mechanical valve • Fontan circulation • Cyanotic heart disease (unrepaired) • Other complex CHD • Marfan syndrome with ascending aorta diameter 40-45 mm • Bicuspid aortic valve syndrome with ascending aorta diameter > 45 mm	Significant increase in risk of maternal mortality or severe morbidity
IV	• Pulmonary arterial hypertension of any cause • Severe systemic ventricular dysfunction (LVEF < 30%) • Previous peripartum cardiomyopathy with any residual impairment of LV function • Severe mitral stenosis (valve area < 1 cm^2) • Severe symptomatic aortic stenosis (valve area < 1 cm^2) • Marfan syndrome with ascending aorta diameter > 45 mm • Bicuspid aortic valve syndrome with ascending aorta diameter 50 mm • NYHA grade III or IV • Native severe coarctation of aorta	Extremely increase risk of maternal mortality or severe morbidity Pregnancy is contraindicated

Abbreviations: ASD = atrial septal defect; CHD = congenital heart disease, LVEF = left ventricular ejection fraction, VHD = valvular heart disease, VSD = ventricular septal defect

Specific valve lesions

1. Mitral stenosis (MS)

Most commonly encountered valvular heart lesion in pregnancy is mitral stenosis, primarily attributed to underlying rheumatic heart disease. Women with moderate or severe mitral stenosis (MS) **(Table 6)** have elevated risks of heart failure and atrial arrhythmias during pregnancy. Conversely, those with mild MS exhibit maternal outcomes similar to individuals without valvular disease. Women with severe MS or symptomatic MS should be reviewed at least monthly.[7]

If diagnosed before conception, women with mild MS should be counselled on minimal risks during pregnancy. On contrary, women with moderate to severe MS should be counselled regarding valvular intervention (percutaneous mitral balloon valvotomy or valve replacement). The main fetal concerns in these women are prematurity (20-30%) and fetal growth restriction (30 %). Severe MS (even asymptomatic) should be counselled against conception and long acting reversible contraception should be advised.

Table 6: Severity of mitral valve stenosis determined by valve area

Severity of mitral valve stenosis	Valve area (cm^2)
Mild	> 1.5
Moderate	1.0 to 1.5
Severe	< 1.0

The medical management of pregnant women with AS who become symptomatic during pregnancy consists of activity restriction, diuresis and digoxin or beta-1 selective agents (metoprolol preferred). Therapeutic anticoagulation

is recommended for women with a prosthetic mechanical valve, severe MS, atrial fibrillation, significant left atrial enlargement, congestive heart failure or having a high risk of stroke. Unresponsive resistant cases need to undergo percutaneous mitral balloon valvotomy (PMBV), which improves the valve area and gradient. The ideal time suggested is before pregnancy or after 20-week gestation due to high risk of ionizing radiations in first trimester. Surgical mitral valve replacement is considered in women with refractory symptoms who are not candidates for PMBV. Surgical interventions have high rates of fetal mortality (20-30%).

2.Aortic stenosis

Aortic Stenosis (AS) is defined as thickening of aortic valve with antegrade velocity across the valve at least 2.0m/sec. If left untreated, leads to worsening of left ventricular hypertrophy. Most common cause of AS is congenital bicuspid aortic valve.When diagnosed prior to conception, proper counselling and evaluation is mandatory. Transthoracic echocardiogram is advised for quantifying the severity of aortic stenosis. Asymptomatic patients are advised to undergo exercise testing so as to evaluate exercise tolerance, arrythmias and need for intervention. Cardiac biomarker NT-proBNP during preconception period indicate severity and risk of complications in pregnancy [7] as well as need for surgical intervention. Risk of heart failure is less than 10 percent and 25 percent in mild -moderate AS & symptomatic severe AS respectively.

Women with mild to moderate AS can safely carry a pregnancy**(Table 7).** On the other hand, women with severe symptomatic AS, reduced exercise test, impaired left ventricular function are advised against pregnancy and are candidates for surgical intervention. Women with moderate to severe AS has 20-25 % chances of preterm birth and fetal growth restriction. Fetal echocardiography is suggested to rule out transmission of congenital cardiac defects.

Table 7: Severity of aortic valve stenosis determined by valve area

Severity of aortic valve stenosis	Valve area (cm^2)
Mild	1.5 - 2.0
Moderate	1.0 to 1.5
Severe	< 1.0

Pregnant women with mild-moderate AS can be reviewed every 3 months antenatally. But women with severe AS in pregnancy should be followed up with cardiologist monthly/bimonthly depending on symptoms. Women who become symptomatic should be managed with activity restriction. Beta-blockers are recommended similarly as in cases of MS. Diuretics should be used cautiously in women who develop pulmonary edema to avoid a sudden drop in preload. In cases where medical treatment is not effective, surgical intervention in the form of percutaneous aortic balloon valvuloplasty is preferred in 2nd trimester.[8] However, if significant aortic regurgitation occurs, percutaneous transcatheter aortic valve replacement may be preferred over valvuloplasty in pregnancy. If surgical intervention is not feasible, then termination of pregnancy or premature delivery with cesarean section followed by valve surgery is advisable.

3. Regurgitant lesions

3a. Mitral regurgitation and aortic regurgitation

Regurgitant lesions are better tolerated than stenotic lesions during pregnancy. The most common reasons for mitral regurgitation (MR) during pregnancy are rheumatic heart disease and mitral valve prolapse. On the other hand, aortic regurgitation (AR) is more frequently associated with congenital bicuspid aortic valve or aortopathy. Even severe MR and AR are well tolerated during pregnancy. Women who experience heart failure symptoms or left ventricular dysfunction can be treated with diuretics and vasodilators, such as hydralazine or nitrates. Surgical intervention before pregnancy is recommended for women with severe symptomatic regurgitant valve disease.In postpartum period, women with regurgitant lesions should be followed closely and diuretics may be added if needed.

3b. Pulmonary regurgitation (PR) is not common during pregnancy and is generally well tolerated. Women with severe PR and underlying right ventricular (RV) dysfunction, RV hypertrophy, or additional obstructive lesions may experience right-sided heart failure. In such cases, activity restriction and diuretics can help manage symptoms. Valve intervention is rarely required during pregnancy, but in women with severe PR who are symptomatic or have progressive RV dilatation or dysfunction, pulmonary valve replacement is recommended before pregnancy.[9]

3c. Pregnant women with **Tricuspid regurgitation (TR)** generally tolerate the hemodynamic changes well, even if the condition is severe. However, women at risk may experience worsening cyanosis and/or arrhythmias during pregnancy. In case of severe TR, symptomatic treatment of right heart failure and atrial arrythmias is advised.[10]

Arrhythmias

Arrhythmias in pregnant women with valvular heart disease can be managed, with anti-arrhythmic agents like flecainide or sotalol, and adenosine or nodal blockade.

Synchronized electrical cardioversion is also considered safe.[11] Anticoagulation is crucial for pregnant women with both mitral stenosis (MS) and atrial fibrillation (AF) due to the increased risk of stroke.

Prosthetic heart valves

The two types of heart valves, bioprosthetic and mechanical valves have unique risks including thrombosis, need of anticoagulation, and durability. Pregnant females with mechanical prosthetic heart valves are at high risk of valve thrombosis, necessitating initiation of anticoagulation therapy. Valve thrombosis is more frequent in first trimester due to inadequate anticoagulation.[13] Bioprosthetic valves have a low risk of thrombosis and don't require anti-coagulation, but have a higher rate of valve deterioration. In case of bioprosthetic valves, low-dose aspirin is advised during pregnancy and an anticoagulant is used only if patient has prior history of thrombosis or atrial fibrillation.

Anticoagulation in women with valvular heart disease

Patient has to be counselled that there is no safe anti-coagulant in pregnancy and shared decision making is recommended.[13] Warfarin, vit-k antagonists (VKA) is considered most effective anticoagulant, but teratogenicity (warfarin embryopathy) remains the major concern with high dose warfarin (30%).

Low molecular weight heparin (LMWH) is an effective alternative to warfarin but carries 10 percent risk of thromboembolic events, especially during switch-over period and presence of sub-therapeutic levels of LMWH. Fixed dose LMWH is associated with higher thromboembolic complications as compared to dose-adjusted regimens. Various factors influencing choice of anti-coagulant are need of high dose of warfarin (> 5mg/day), type of valve and thrombogenicity, compliance, availability of LMWH monitoring (anti-Xa levels) and history of thrombotic events. American Heart Association (AHA) 2020 recommended simultaneous use of low dose aspirin in second and third trimester, although ESC (European society of cardiology) guidelines 2018 does not recommend the use of aspirin.

The three most commonly used anticoagulation strategies include

- Vit-K antagonists (warfarin) throughout the pregnancy – switch to LMWH twice daily is made 1-2 weeks before expected delivery.
- LMWH in first trimester and VKA in second and third trimester – Patient continues taking VKA till 6th week of gestation. Switch over to LMWH is done at 6th week and LMWH is stopped at 12th week of gestation.Then VKAs are again started at 12th week and continued in second and third trimester. The switchover to LMWH is again done prior to delivery.
- LMWH throughout pregnancy – When pregnancy is confirmed, switch to LMWH twice daily is made. LMWH is continued till delivery.

If there is non-availability of LMWH or monitoring, IV unfractionated heparin (UFH) can be considered as a safe alternative. Anticoagulation is interrupted 4-6 hours before delivery.

Antenatal management

Close clinical monitoring with echocardiography once every trimester and 3rd trimester echocardiography to be performed around 32 weeks is recommended. Fetal echocardiography at around 18 to 22 weeks is recommended in pregnant females with congenital heart diseases. Serial ultrasonography to rule out fetal growth restriction is also recommended.

Labor and delivery

Vaginal delivery is preferred in cases of pregnant women with VHD with mild symptoms and not on therapeutic anti-coagulation. However, in moderate to severe left-sided obstructive lesions second stage of labor should be shortened using forceps or ventouse. Regional anesthesia during vaginal delivery can reduce stress on the heart. Epidural is preferred over spinal anesthesia due to its lesser association with hypotension. IV fluids should be used to maintain euvolemia.[14]

If a woman on warfarin goes into labor or there is need for urgent delivery, the cesarean section should be performed to avoid intracerebral hemorrhage in neonate.[13] Cesarean section may be necessary for women with severe heart failure (NYHA class III &IV) or high-risk aortic disease, or pulmonary hypertension.

Post-partum Period

Post-delivery close monitoring of hemodynamics and volume status in an intensive care unit is recommended, especially in severe valvular heart disease because of severe third space shifts which occur in post-partum period. Postpartum hemorrhage remains a major challenge to be dealt in these patients. Multidisciplinary decision to balance risk of PPH vs thrombosis is required. If patient has undergone vaginal delivery with no neuraxial catheter, restart anticoagulation with LMWH after 6 hours. If the neuraxial catheter is used or in case of cesarean section, anti-coagulation has to be started after 12 hours. Warfarin is restarted on fifth postpartum day.[13]

Family planning

In situations, where the couple is certain about completion of family, sterilisation at time of elective cesarean or vasectomy in male partner may be considered appropriate. Oral combined contraceptive should be avoided due to increased risk of thrombosis.[14] Long-acting progesterones (injections & implants) are considered safe. Intra-uterine copper devices and levonorgestrel containing devices are also very effective. [15]

Lactation

There is no contraindication for breast feeding even in patients on warfarin, as inactive metabolite of warfarin is excreted in breast milk.

Conclusion

Valvular heart disease during pregnancy requires multidisciplinary and individualised care in a well equipped centre. Timely pre conceptional counselling, proper anticoagulation therapy, appropriate surgical interventions, due considerations during labor, delivery and postpartum period are the pearls for achieving low maternal morbidity and mortality.

Key points

- The physiological changes that occur during pregnancy can significantly impact the cardiac function of women with pre-existing valvular disease.
- In pregnancy, regurgitant valve lesions are better tolerated as compared to stenotic lesions, though both carry an increased risk of obstetric complications.
- Pre-conceptional counselling is must for women with VHD.
- Ensuring optimal anticoagulation poses a unique challenge for women with mechanical valves.
- Vaginal delivery with epidural anaesthesia is recommended in most patients of stable valvular heart disease and the cesarean section is considered for obstetrics indications and severe VHD.
- Long acting reversible contraceptives (IUD, injectables, implants) are considered safe in women with VHD.

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> Motherhood is a series of behaviors rather than a role for heroes.
>
> *–Margaret Wheatley*

An Update of Medical Nutrition Therapy in Gestational Diabetes Mellitus

Dr Ragini Agrawal, M.S.
A.A. DERMASCIENCE "Complete Clinic for Women – HER LIFE Matters", Gurugram
9711187176, drraginiagrawal@gmail.com

Gestational diabetes mellitus (GDM) is a serious and frequent pregnancy complication that can lead to short and long-term risks for both mother and fetus.Different health organizations proposed different algorithms for the screening, diagnosis, and management of GDM.

GDM is operationally defined as impaired glucose tolerance with onset or first recognition during pregnancy. In accordance to World Health Organization recommendations, the GOI guideline endorses 2-h 75-g OGTT, irrespective of last meal timings with a cutoff value of e"140 mg/dL using a plasma-standardized glucometer. If woman tested positive (2-h 75-g OGTT e"140 mg/dL), she is managed as per GDM protocols. However, if tested negative, OGTT is repeated at 24–28 weeks ensuring a minimum gap of at least 4 weeks from the first test.

Clinical Management

GDM is managed through MNT along with physical activity, followed by subsequent 2-hr PPBS testing at 2 weeks which is maintained below <120 mg/dL.

If 2-h PPBS remains e"120 mg/dL, medical therapy (insulin/metformin) is added to MNT as per the guidelines

Milestone for GDM treatment in order to reduce maternal and fetal complications

How to achieve the goal

All these interventions involve a strong collaboration between the pregnant woman and the medical care team, based on "Mutual trust and correct information" .A sustained psychosocial support represents an important part of the therapy.

Steps to successful MNT

1. MNT, together with physical exercise, weight control, and implementing a self-control strategy, should begin as soon as possible after diagnosis, namely, in the first week.
2. Pregnant women should be taught to self-monitor fasting and postprandial glucose and to keep a diary where to note down the values of self-measured blood glucose, data on the food & physical exercise,
3. Diary should be presented to the medical team. Diary will help physician
   - To identify the individual variations of glycemic values and the factors determining them
   - Taking appropriate decisions regarding their lifestyle changes.

The glycemic targets recommended for pregnant women with GDM are as follows

Time of monitoring	Value
FBS	< 95 mg/dl
PP 1 hr	< 140 MG/DL
PP 2hr	**< 12 MG /DL**

It was shown that reaching and maintaining fasting 95 mg/dl in the first 2 weeks from implementing MNT are correlated with a reduced possibility of introducing pharmacological treatment.

MNT in GDM has the following main objectives:

Providing the appropriate caloric intake for both mother and fetus

- Providing the appropriate caloric intake for both mother and fetus
- Promoting optimal fetal growth
- Avoiding ketosis
- Avoiding the mother's excessive weight gain.

The nutrition plan is individualized, taking into consideration the mother's particularities (health state, weight, ethnic, cultural particularities, compliance, etc.). The medical team being the one informing the mother about the risks that this condition may have upon her and the fetus, in order to obtain maximum compliance and adherence.

MNT is of utmost importance for the management of this condition as Studies showed that 70-85% of pregnant women diagnosed with GDM obtained and maintained glycemic targets only with MNT.

Caloric Intake

Weight loss during pregnancy is not recommend .If the caloric restriction is required, this should be performed in a controlled manner, taking into consideration the fact that severe food restriction may lead to a rapid turn of the body into using fatty acids (FA) and glucose saving,Negative effect of maternal ketonemia, is being associated with neurological disorders and future cognitive deficits in the baby

Maternal hyperglycemia induces an excess of nutrients in the fetal blood stream which leads, through multiple mechanisms to fetal macrosomia and its multiple complications like

1. Mechanical complications during delivery
2. Obesity, and diabetes during the teenage period or adulthood

As high percentage of overweight and obese women at their reproductive age (25-40%)

"It is very important to intervene on women's lifestyle, conducting information campaigns, and aggressively fight obesity before conception, in order to provide a healthy start in life for future generations"

Regarding overweight and obese women, moderate caloric restriction is indicated (a reduction by approx. 30% of the caloric intake prior to pregnancy, taking into consideration that the diet should not have under 1600 kcal/day)

The caloric intake of pregnant women with GDM

BMI prior to pregnancy (kg/m²)	Caloric intake (kcals/kg/day)
< 18.5 (underweight)	35–40
18.5–24.9 (normal weight)	30–34
25–29.9 (overweight)	25–29
≥ 30 (obesity)	Maximum 24 kcals/kg/day or a reduction of 30–33% of the prior caloric intake

GOI Guidelines

- Energy requirement does not increase in the first trimester unless a woman is underweight.
- Energy requirement increases during second and third trimester.
- Energy intake should be adequate enough to provide appropriate weight gain during pregnancy.
- As per Indian ICMR guidelines for an average weight gain of 10- 12 Kg, an addition of 350 K.cal/day above the adult requirement is recommended during second and third trimester.

Equations proposed by ICMR (1989) expert group can be used to calculate adult energy requirement which are as follows:

- Energy requirement (K.cal/d) = BMR × PAL
- *BMR = Basal metabolic rate *PAL = Physical activity level
- BMR (K.cal/d)for adult females
- (18-30yrs) = 14 × B.W (Kg) + 471
- l BMR (K.cal/d)for adult females (30-60yrs) = 8.3 × B.W (Kg) + 788 * (B.W = body weight)

PAL values proposed by ICMR expert group (2009) are as follows

Level of activity	PAL value
Sedentary work	1.53
Moderate work	1.8
Heavy work	2.3

Pre-pregnancy weight BMI (kg/m2)	Total weight gain range (kg)
Normal weight 18.5 to 24.9	11.5 to 16 kg
Under weight Less than 18.5	12.5 to 18 kg
Over weight 25 to 29	9 7 to 11.5 kg
Obese (include all classes namely grade I, II, and III) Equal/more than 30 5 to 9 kg	5 to 9 kg

Carbohydrate Intake

The idea of carbohydrate (CH) dietary restriction in GDM has its origin even before the insulin era, when it was noted that a severe restriction of CH (8-10% of the total caloric intake) prolonged life in women with type 1 diabetes and reduced the incidence of fetal macrosomia and stillbirth. Numerous studies correlated maternal hyperglycemia with fetal macrosomia.

In 1990, Jovanovic-Peterson and Peterson proposed that the CH restriction should be considered the first line of treatment in GDM. In the following decades, there was an emphasis on identifying the most appropriate type of diet that provided optimal results, both for the mother and for the fetus.

There are numerous controversies regarding the optimal intake of CH, in terms of quantity and type of CH .It raises the question whether the best approach is represented by the CH restriction or by a more "liberal" diet. In the last decades, the emphasis went more and more on the use of low glycemic index (GI) CH. The glycemic index is a value assigned to foods that defines their impact on postprandial glucose values .Complex starch with low glycemic index should be preferred.

Low GI (<55)	Medium GI (55-69)	High GI (70-100)
Cauliflower, leek, cabbage, beans, strawberries, peaches, apples, plums, pineapple, milk, yogurt, rye bread, whole grain pasta	Bananas, jam, honey, couscous, pizza, polenta, whole flour bread	Chocolate, donuts, potatoes, white flour, corn flakes

ADA recommendations are that pregnant women with GDM should consume a minimum quantity of 175 g CH/day, representing 35-50% of the total caloric intake.

The quantity and distribution of CH should be made according to the particularities of every pregnant woman:

BMI

Weight gain during pregnancy,

fasting and postprandial glucose values

Presence or absence of ketonemia.

Most guidelines recommend the distribution of CH into

A. 3 main meals

- Breakfast: 10-15%,
- Lunch: 20-30%
- Dinner: 30-40%

B. 3 small snacks

(5-10% of the total CH intake)

The CH intake during breakfast should be reduced to 15-30 g, taking into consideration the morning peak of cortisol secretion, which explains why most pregnant women with GDM present high blood glucose values after breakfast.

Numerous studies suggest that diets based on food with a low GI improve the glycemic profile of mothers with GDM and reduce the risk of fetal macrosomia.

This aspect could also be used in deciding a menu for breakfast, especially in women who have difficulties in controlling postprandial glucose during this time of the day

Fiber Intake

ADA recommends an intake of **28 g/day,** coming mainly from cereals, fruits, and vegetables, due to their well-known positive effect on the control of postprandial glucose.

Studies that investigated the effect of high fiber intake diets (80 g/day) reported a low compliance of pregnant women to this type of diet (40-60%), due to the gastrointestinal side effects. A Meta-analysis highlighted that the risk for fetal macrosomia was reduced in pregnant women with GDM who had a diet based on low GI foods and high fiber intake, in comparison to those having diet with low GI foods and low fiber intake

Protein Intake

During pregnancy, an appropriate protein intake is crucial in order to promote fetal growth and development. There is no evidence from studies indicating a particularity of pregnant women with GDM, neither regarding the protein quantity recommended during pregnancy nor their type.

- ADA recommends a protein intake of a minimum 71 g/day in pregnant women with GDM for all stages of pregnancy
- Recommendations regarding the protein and amino acid intake should vary according to the gestational age, in order to adequately fulfill the increasing needs of the mother and the fetus
- Protein requirements to increase from 1.2 g/kg/day at 16 weeks of pregnancy to 1.52 g/kg/day at 36 weeks

The main protein sources are represented by low-fat white and red meat, eggs, soya, nuts, and vegetables. Animal products should be very well and healthy cooked. Fish and seafood represent an extremely rich source of proteins, iron, and omega-3, vital for the development of the fetus brain.

Special Remarks for sea food

These species commonly come from mercury-polluted

water, this leading to intoxications, with serious effects on the mother and fetus (neurological damage, cognitive, attention, memory, and language problems

Lipid Intake

Tendency of pregnant women to consume a higher quantity of lipids was shown in numerous studies to have negative consequences for the health of both mother and fetus.

1. High level of free fatty acids (FFA) increases insulin resistance.
2. High level of triglycerides (TG) and FFA in the maternal serum was correlated with fetal macrosomia, due to TG hydrolysis and the FFA transport through the placenta to the fetus where it contributes to an excessive fetal growth.

A study performed on 34 pregnant women following the diet DASH (Dietary Approaches to Stop Hypertension) (65% CH, 18% lipids) for 4 weeks highlighted the following beneficial effects: decrease of glycated hemoglobin (HbA1c), of systolic blood pressure, of seric lipids, and of oxidative stress and improvement of insulin resistance. At present, recommendations indicate a lipid intake of 20-35% of the total caloric intake.

The saturated FA and Trans FA should be reduced as much as possible, down to 7% from the caloric intake. As such, pregnant women are advised to choose meat with a fat content below 10%, as well as low-fat dairy products. The remaining percentage is divided between

- **Monounsaturated fatty acids (MUFA) (olive oil, nuts, peanut, nuts, and avocado),**
- **Polyunsaturated fatty acids (PUFA), omega-3 (fish, fish oil, and flax oil)**
- **Omega-6 (soya oil, sunflower, rape, and corn oil).**

The report between these three types of FA is not clearly defined in pregnant women with GDM.

There were studies highlighting the fact that supplementing the diet with PUFA n-3 reduced fetal macrosomia

Recommended carbohydrate, protein, and lipid intake in GDM.

Macronutrients	% caloric intake
Carbohydrates	35–50% (minimum 175 g/day) (ADA)
Proteins	71 g/day (ADA)
Lipids	20–35% (IOM)30–35% (DDG)

Vitamin and Mineral Intake

Pregnancy represents a time when women need a high intake of vitamins and minerals, in order to ensure both their needs and the ones of their babies. In a varied and correct diet, all their needs should be covered.

Folic acid is essential for the synthesis of nucleic acid, being vital for fetal growth. Supplementing the diet with folic acid before conception and during the first 12 weeks of pregnancy considerably reduced the percentage of pregnancies with neuronal tube defects in children. Folic acid are recommended in a dose of 5mg/day, 3 months before conception, reducing the dose down to 0.4-1 mg/day starting from the 12th week of gestation

Vitamins C and E are known as strong antioxidants and are very important in the diet of all pregnant women, being the well-known fetotoxic role of the oxidative stress.(No RDA)

Recently, a meta-analysis that collected data from the studies performed on a total of 249 975 pregnant women showed a clear relation between the administration of supplements with vitamin D and multivitamins and the reduction of the risk for preeclampsia.

A recent meta-analysis that included 6 randomized studies concluded that the administration of vitamin D supplements led to the improvement of insulin sensitivity, still not to the reduction of fasting glucose or HbA1c.

Recommended dose —5 µg/day -10µg/day

During pregnancy, supplements with vitamin A are contraindicated.

The calcium necessary is high during pregnancy.At present, an intake of 900-1000 mg calcium/day is recommended

Iron

According to CDC the iron necessary during pregnancy is of 27 mg/day. This may be ensured through a correct diet, iron supplements being required only in the case of an iron-deficiency anemia. The results of a prospective study performed on 3 158 pregnant women identified a 50% higher risk for GDM in pregnant women who had an excess of heme iron (mainly found in chicken meat and red meat)

Regarding the intake of coffee, alcohol, and smoking, pregnant women with GDM are to follow the general recommendations during pregnancy: alcohol is strictly prohibited (risk for fetal alcoholic syndrome), caffeine intake should be reduced to a maximum of 200 mg/day, and smoking should be discouraged.

Caffeine content of different beverages:

Drink	Average amount of caffeine (mg)
Brewed coffee 220 ml	135 (80-200)
Instant coffee 220 ml	75
Instant tea 220 ml	26-36
Soft drinks (Cola) 330 ml	35

New Research Directions for GDM Prevention

Plant-Based Diets

Scientific evidence suggests that plant-based diets can prevent type 2 diabetes by decreasing gastric emptying, improving insulin sensitivity, and increasing insulin secretion.

Lately, there are new evidences that suggest that a diet based on plant-derived food may have a positive impact on GDM also by enhancing antioxidant compounds

- Women with GDM have increased levels of oxidative stress and inflammatory markers (tumor necrosis factor alpha (TNF-á), interleukin 6 (IL-6), and C-reactive protein (CRP)) that could be modulated by diets based on plant-derived food such as the Mediterranean Diet.
- This diet is based on: vegetables, fruits, nuts, seeds, oils, beans, and whole grains.
- Taking into consideration the role that some cytokines, especially IL-6, play in the respiratory syndrome of COVID-19 and the fact that the Mediterranean Diet can modulate TNF-á, IL6, and CRP, there are reasons to believe that this diet can reduce the risk of GDM and improve the immune response in COVID-19 pneumonia

Myo-inositol

The reduction of GDM incidence was quoted by numerous other studies performed on pregnant women with obesity/overweight/family history of DM or glucidic metabolism change, all these results being in favor for the administration of myo-inositol supplements in women at risk, in order to prevent GDM

To conclude reaching an international consensus regarding the screening, management, and follow-up for women with GDM is of extreme importance in order to prevent the short and long-time complications. Women with GDM should receive MNT as soon as possible after the diagnosis, but prevention is of utmost importance among pregnant women and women that are trying to conceive.

Pregnancy represents a special time in the life of every family, and it should be seen as an opportunity to implement a healthy lifestyle and to break the vicious circle of unhealthy choices and obesity and metabolic syndrome transmitted from one generation to another. In this period of time, families are more motivated and committed to changes and healthy choices. This is the right moment when the medical team may implement efficient prevention strategies for fighting against the epidemics of obesity and DM.

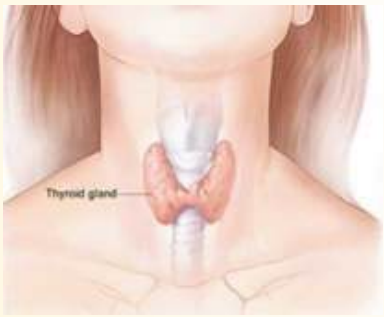
> The influence of a mother in the lives of her children is beyond calculation.
>
> *–James E. Faust*

Thyroid Disorders In Pregnancy

Dr Taru Chhaya
MS OBGY

Introduction:

Thyroid disorders in pregnancy can have impact on both maternal and fetal health. This article is to provide a comprehensive overview of the impact, management and implications of thyroid disorders in pregnancy. Here we will discuss prevalence, Pathophysiology, clinical manifestations and diagnostic criteria for hypothyroidism and hyperthyroidism in pregnant women. Additionally we will discuss complications and their prevention by management and diagnostic strategies.



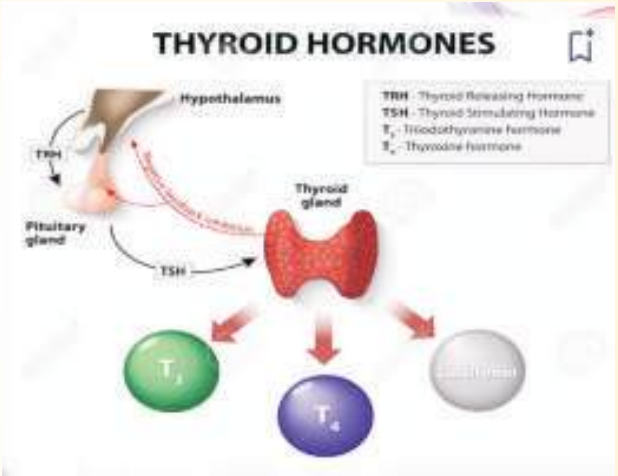
A normal pregnancy results in many reversible physiological and hormonal changes and one of them is Thyroid disorder.

Definition:

Pregnancy is a time of complex hormonal changes. Thyroid disorders are encountered frequently during pregnancy. Maternal hypothyroidism in pregnancy refers to any state in which Thyroid hormone production is below normal during pregnancy and Hyperthyroidism when T3and T4 are higher than normal.

Normal Physiology:

The thyroid gland, anterior pituitary gland, and hypothalamus comprise a self-regulatory circuit called the hypothalamic-pituitary-thyroid axis. Thyrotropin-releasing hormone (TRH) is released from the hypothalamus and thyroid-stimulating hormone (TSH) from the anterior pituitary gland. The main hormones produced by the thyroid gland are thyroxine or tetraiodothyronine (T4) and triiodothyronine (T3). TRH, TSH and T4 work in synchronous harmony to maintain proper feedback mechanisms and homeostasis.



Prevelance:

Approximately 2-3% of pregnant women experience hypothyroidism and 0.2-0.3% experience hyperthyroidism.

The incidence of overt hypothyroidism is 0.2-2.5 % and that of subclinical hypothyroidism is 2-7%.

Infact thyroid antibodies are present in almost 60% of reproductive age women.

Changes During Pregnancy:

Pregnancy has a profound impact on the thyroid gland and its function. During pregnancy, the thyroid gland increases in size by 10% in iodine replete countries but by 20% to 40% in areas of iodine deficiency. Production of the thyroid hormones, thyroxine (T4), and triiodothyronine (T3), increases by nearly 50%, in conjunction with a separate 50% increase in the daily iodine requirement.

HCG released by placenta has structural similarities with

TSH. During pregnancy high HCG stimulates thyroid gland to secrete more thyroxine (T_4) and triiodothyronine (T_3) which in turn by feedback mechanism inhibit TSH secretion. TSH should be low but in case we find higher value it is considered as Hypothyroidism.

GUIDELINES:

According to American Thyroid Association (ATA) guidelines 2011:

The recommended reference ranges for TSH are

0.1 to 2.5 mIU/L in the first trimester,

0.2 to 3.0 mIU/L in the second trimester, and

0.3 to 3.0 mIU/L in the third trimester.

Guidelines 2012 India ·

1st : 2.5 mIU/L · 2nd : 3.0mIU/L · 3rd : 3.0 mIU/L

Guidelines 2014 European ·

1st : 2.5 mIU/L · 2nd : 3.0mIU/L · 3rd : 3.0 mIU/L

Types Of Thyroid Disorders In Pregnancy:

Hypothyroidism:

This is the condition when thyroid function is less than normal.

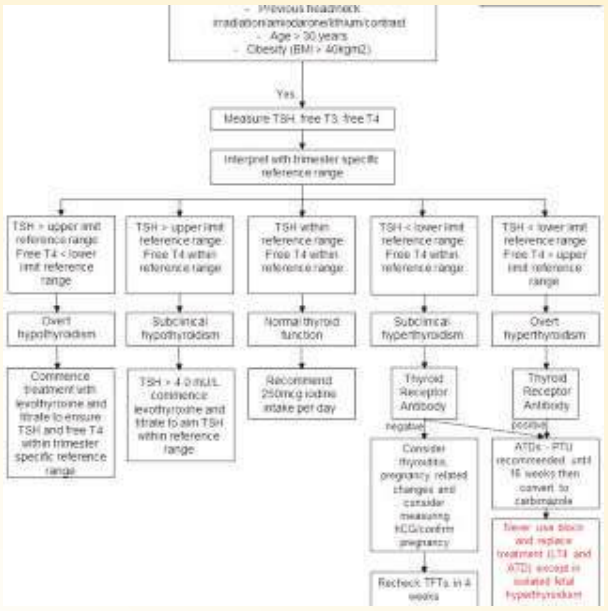
Subclinical Hypothyroidism subclinical hypothyroidism is serum TSH level in the of 4-10 mIU/L with normal thyroxine (T4) level.

Overt Hypothyroidism Overt hypothyroidism is increased in serum TSH (more than 10 mIU/L) as a result of decrease thyroxine and a negative feedback

Diagnostic Criteria: Screening for thyroid disorders is typically recommended during early pregnancy or at the first prenatal visit, particularly in high-risk individuals.

The primary screening test is serum Thyroid Stimulating Hormone (TSH) measurement, with additional testing for free thyroxine (F T4) and thyroid peroxidase antibodies (TPOAb) in specific cases.

Trimester-specific reference ranges for TSH levels should be considered, as they vary throughout pregnancy.



Sign And Symptoms:

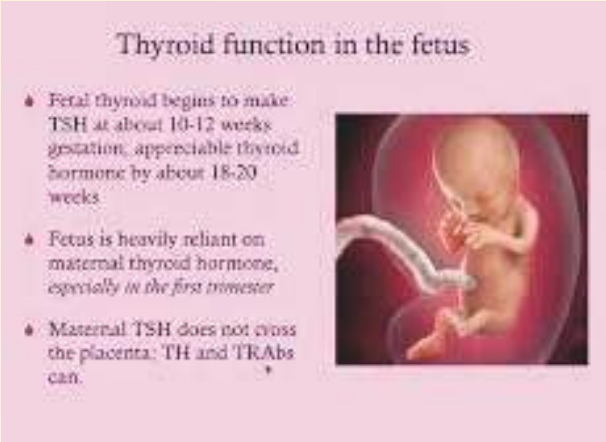
- Edema all over
- Hair loss
- Puffiness of face
- Lethargy
- Forgetfulness
- Constipation
- Cold Intolerance
- Insomnia

Effects Of Hypothyroidism In Pregnancy:

- Maternal Risks:
- Anaemia and CHF
- Pre-eclampsia
- Placental abnormalities
- Post partum hemorrhage
- Myopathy
- Hyperemesis
- RPL
- Premature Delivery
- Cognitive impairment

Fetal Risks:

- Neurological abnormalities
- Developmental abnormalities
- Low Birth Weight



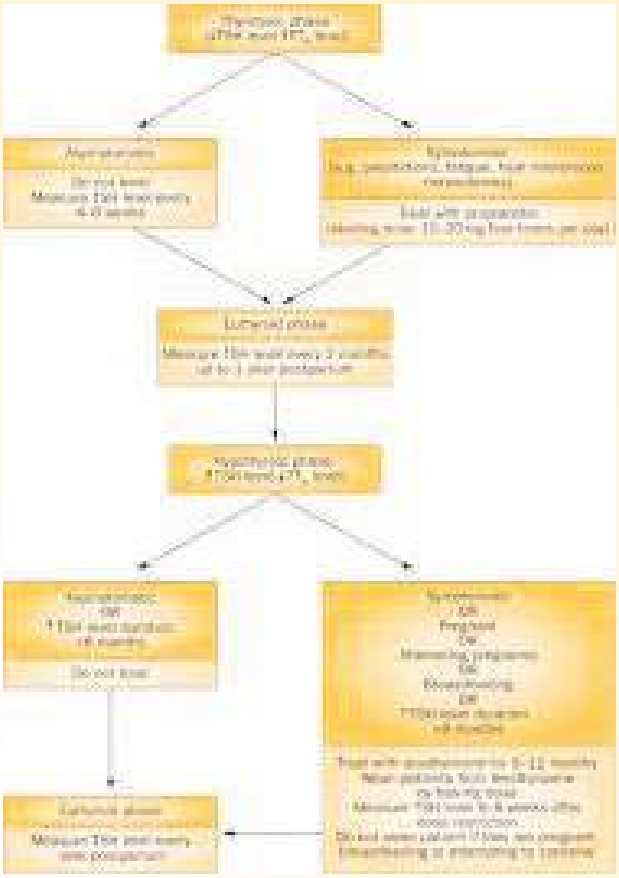
Neonatal Risks:

- Hyperbilirubinemia
- Respiratory distress
- Delayed Milestones
- Congenital Hypothyroidism

Management:

FOGSI 2019 Recommendations For The Management Of Thyroid Dysfunction In Pregnancy

- During Pregnancy, all patients with overt hypothyroidism with TSH > 2.5 mIU/L should be treated with Levothyroxine (LT4) dose 1.6-2.0 ìg/kg/day.
- The target of TSH levels d"2.5 mIU/L should be maintained.
- In patients with pre-existing hypothyroidism, Levothyroxine(LT4) dose increased by 30% as soon as pregnancy is diagnosed.
- Regular TSH monitoring (approximately every 4-6 weeks until 20 weeks gestation and at least once near 28 weeks gestation) should be done.
- LT4 therapy is recommended for women with a TSH greater than 10.0 mIU/L and TPOAb positive women with a TSH between 4-10 mIU/L.
- LT4 therapy can be considered for women with TPOAb negative with a TSH between 4-10.0 mU/L and TPOAb positive women with a TSH between 2.5-4 mU/L.



Nutrition and Diet

Iodine rich foods... Spinach, Figs, Almonds, Cardamom, Quinoa, Kidney beans, Soyabeans

Green vegetables... Spinach, Cabbage, Peas, Bell pepper, Legumes

Zinc rich foods... Pumpin, Mushroom, Wheat germ, Sesame seeds, Cereals, Meat

Management Of Hypothyroidism Post-partum And Follow-up:

Post-delivery the patient should be reverted back to the pre pregnant dosage and TSH levels should be rechecked after 6 weeks.

Some women in whom LT4 is initiated during pregnancy may not require LT4 postpartum. Such women are candidates for discontinuing LT4, especially when the LT4 dose is d"50 mcg daily.

If LT4 is discontinued, serum TSH should be evaluated in 6 weeks.

Women with thyroid autoimmunity need annual monitoring with TSH.

Hyperthyroidism:

Definition: Overproduction of Thyroid hormones during pregnancy

Prevalence: It affects 2/1000 pregnancies

Diagnosis: Low TSH with elevated T4, F T4 and F T3

Causes:

1. **Autoimmune Hyperthyroidism (Grave's Disease)** Fetal Graves' disease is very rare and happens due to trans placental transfer of TSH-receptor stimulating antibody.
2. Functional Adenoma
3. Sub acute Thyroiditis
4. Trophoblastic diseases
5. Hyperemesis Gravidarum

Signs and Symptoms:

- Nausea and Vomiting
- Weight loss
- Breathlessness
- Nervousness
- Heat intolerance.
- Resting Pulse > 100/mt
- Palpitation
- Diffuse Goiter
- Increased cardiac output

Maternal Risk:

- Preterm Delivery
- Early Pregnancy loss
- Pre Eclampsia
- Infections
- CHF

Transient gestational thyrotoxicosis, where free thyroid hormone can be increased, and they require a short course of anti-thyroid medication and recovers over a period of few weeks.

Untreated mothers can also develop thyrotoxic crisis, fortunately rare now with an early diagnosis.

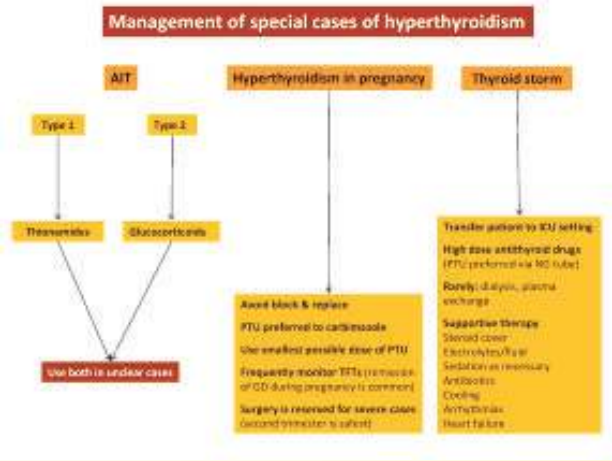
Fetal Risk:

- IUGR
- Low birth weight baby
- Stillbirth
- Hyperthyroidism
- Increased perinatal morbidity.

Management:

Medical...

- Control of symptoms.
- The preferred regimen is titration regimen.
- Preferred medicine is Propylthiouracil (PTU). The dose of PTU depends on the control, sometime goes even up to 400-800 mg/day. It is to be given every 8 hourly.
- Liver function tests should be monitored with PTU, as there is a risk of hepatotoxicity.
- Methimazole is not preferred in the first trimester due to the risk of aplasia cutis and the spectrum of birth defects in pregnancy.
- Methimazole can be given in the second and third trimesters.
- The aim is to keep free T4 in the upper normal range
- Sometime TSH can be little lower than normal range.
- Free T4 is more reliable as TSH takes time to get settled.
- **Block and replace regimen** is not followed in pregnancy as thyroxine does not cross placenta freely but anti-thyroid medications do.



Surgical:

- Subtotal Thyroidectomy
- Thyroidectomy

PRE and POST pregnancy management:

- Hyperthyroidism if diagnosed before conception is best treated before conception. In case radioactive iodine is given, current recommendation is not to conceive for at least four months.
- Monitoring should be close as patient may get recurrence or aggravation of symptoms in first few months postnatal.
- Check TSH and T4 every 6 weeks
- Breast feeding has no issues as most medications are protein bound.

Take Home Message:

- Thyroid disorders are common in pregnancy.
- The most common disorder is subclinical hypothyroidism.
- Early and effective treatment of thyroid disorder ensures a safe pregnancy with minimal maternal and neonatal complications.

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"When you are looking at your mother, you are looking at the purest love you will ever know."

–Charley Benetto

"Motherhood has a very humanizing effect. Everything gets reduced to essentials."

–Meryl Streep

Jaundice in Pregnancy

Dr Seema Mehrotra
Professor,Dept Of OB&GYN
KGMU, Lucknow
mehrotra.seema@gmail.com

Dr Nikita Kausal
Senior Resident, Dept Of OB&GYN
KGMU,Lucknow
nikitakausal401@gmail.com

Introduction

Liver disease during pregnancy can complicate up to 3% of all pregnancies[1].In India incidence of jaundice in pregnancy varies from 0.4 to 0.9/1000 deliveries[2]. During pregnancy liver diseases can lead to high maternal and fetal morbidity and mortality.

The signs and symptoms of liver disease in pregnancy are often non specific and consist of jaundice, nausea, vomiting and abdominal pain[3].

The occurrence of hepatobiliary disease with or without jaundice during pregnancy provides interesting and urgent diagnostic challenge to obstetrician and hepatologist. Advances in understanding and management of liver disorders unique to pregnancy and hepatobiliary disease in general have resulted in a significant improvement in maternal and fetal outcome.

Liver In Normal Pregnancy

In pregnancy plasma volume increases by 50%, but with disproportionate increase in red cell mass by 20%, there is resultant hemodilution. This phenomenon should be kept in mind during interpretation of all serum concentrations used in evaluation of hepatic function during pregnancy. Serum albumin decreases, serum cholesterol, triglyceride and fibrinogen increases. Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), prothrombin (PT), bile acid levels are not affected. Serum alkaline phosphatase level almost doubles due to its placental isozyme bilirubin, gamma glutamyltranspeptidase(GGTP) slightly decreases.

Liver disorders that occur in pregnancy can be divided into three groups:

1 **Liver diseases unique to pregnancy**
- Hyperemesis gravidarum
- Intrahepatic cholestasis of pregnancy
- In pre eclampsia and eclampsia, HELLP syndrome
- Acute fatty liver of pregnancy.

2 **Liver diseases coincidental to pregnancy**
- Viral hepatitis A, B, C, D, E herpes simplex
- Malaria, sickle cell crisis, leptospirosis
- Gallstones, Budd-Chiari syndrome
- Drugs.

3 **Pregnancy in preexisting liver disease**
- Chronic viral hepatitis, hepatitis B virus (HBV), hepatitis C virus (HCV), nonalcoholic fatty liver disease
- Portal hypertension, autoimmune hepatitis, Wilson's disease
- Primary biliary cirrhosis, primary sclerosing cholangitis.
- Liver tumors.[4,5]

Liver disorders may present themselves inconspicuously during gestation. Due to the physiological changes in the liver that occur during pregnancy, it is difficult to diagnose and manage a liver disease.

A systematic approach is needed for its treatment and must include a detailed clinical history, examination, laboratory analyses and radiographic evaluation. The clinical history should include duration of jaundice, interval between symptoms and jaundice previous pregnancy diseases and associated liver complications, intravenous drug use, transfusions, if pregnancies were taken to term, and oral contraceptive use Exposure to contaminated food or water, Family history of hemolytic anemia, sickle cell disease, congenital hyperbilirubinemia Also, the patient should be evaluated for clinical data that may suggest liver dysfunction such as nausea, vomiting, jaundice, generalized pruritus, abdominal pain, polyuria and polydipsia in the absence of other morbidities as chronic metabolic disease such as diabetes[6,7,8,9,10]

Investigations

- Complete blood count [hemoglobin (Hb), total count (TC), differential unit (DC), platelet count, indices]
- Blood group, blood sugar, peripheral smear
- Liver function tests: ALT, AST, serum bilirubin, alkaline phosphatase, serum albumin, and globulin, lactate dehydrogenase (marker of hemolysis) elevated >600 U/L in HELLP syndrome, gamma-glutamyl transferase (GGT)
- Renal function tests: Urine—routine and microscopy, serum urea, uric acid, creatinine x Coagulation profile: Prothrombin time (PT), activated partial thromboplastin time (APTT), serum fibrinogen, fibrin degradation product (FDP), D-diamer
- Serum electrolytes x IgM HAV (hepatitis A virus)
- HBsAg (hepatitis B virus) x Antibodies and PCR tests for HCV (hepatitis C virus)
- IgM HEV (hepatitis E virus)
- Tests for HSV (herpes simplex virus)
- USG, CT scan for hemorrhage in liver, MRI, liver biopsy rarely
- Tests for fetal surveillance—electronic fetal monitoring, ultrasonography, color Doppler.

Liver Disease Unique to Pregnancy

Hyperemesis gravidarum is persistent nausea and vomiting associated with 5% or greater weight loss,fluid and electrolyte imbalance, dehydration and ketonuria. Incidence varies from 0.3 to 2% of live births[11].It usually occurs during first trimester and resolves by 20th week of gestation.It occurs due to increase in serum levels of chorionic gonadotropin or estrogen or overactivity of hypothalamic-pitutitary-adrenal axis.Jaundice induced by hyperemesis is caused by conjugated hyperbilirubinemia. Bilirubin levels may be normal or increased.Serum transferase activity is elevated but <500IU/L[2].

Treatment is supportive which includes correction of dehydration and electrolyte imbalance,antiemetics,folate and thiamine supplements. Patient should eat small, frequent, low fat meals.

Intrahepatic cholestasis of pregnancy also known as icteus gravidarum/obstetrics cholestasis/recurrent jaundice of pregnancy.It is the second most common cause of jaundice in pregnancy after viral hepatitis.It affects 0.7% of pregnancies in UK and 1.2-1.5% of women of Indian-Asian or Pakistani-Asian origin[6].ICP is characterised by pruritis in absence of primary skin condition with elevated maternal serum bile acids in late second or third trimester. It has multifactorial etiology which includes genetic, hormonal and environmental factors. Pregnancy associated increase in estrogen,progesterone and mutation in canalicular bile salt export pump(ABCB11) and MDR3, ABCB4 gene which impairs transportation of bile acids. Risk factors include multiparity, advanced maternal age,history of cholestasis with OCP use and chronic hepatitis C. The clinical manifestation includes unexplained pruritis which usually begins in palms and soles and may progress to rest of the body,often worsens at nights. Jaundice may occur in approximately 10% of patients. Other symptoms may include fatigue, anorexia, epigastric pain, steatorrhoea.

The diagnosis of ICP includes itching in skin of normal appearance and raised peak random total bile acid concentration of 19 micromol/L or more.Bilirubin levels may be increased but usually less than 5 mg/dl,alkaline phosphatase elevated more than that in normal pregnancy.

Fetal complications may includes preterm delivery, meconium staining of liquor, stillbirth due to fetal arrhythmia.

Ursodeoxycholic acid (UDCA)is superior to other medications like cholestyramine, S-adenosyl-L-methionine. UDCA is bile salt chelating agent and reduces maternal itching and improves liver test abnormalities.

ICP usually resolves after delivery but in rare cases it may persist after delivery leading to fibrosis or even cirrhosis.

HELLP Syndrome ischaracterised by hemolysis(H), elevated liver enzymes (EL),and low platelet count(LP). Incidence is 0.5-0.9% among all pregnant women and 10-20% in preeclampsia and eclampsia cases[9]. It need not to be accompanied by severe hypertension or proteinuria. Most of the cases usually present between 27-37weeks of gestation while 25-30% cases in postpartum period.

It is a microangiopathic hemolytic anemia associated with fibrin deposition in blood vessels and platelet activation with platelet consumption.

Early diagnosis of HELLP is critical because of high morbidity and mortality rates associated with it.Disseminated intramuscular coagulation (DIC) may be present.Aminotranferase levels may increase to 10-20 fold,bilirubin levels <5mg/dl. PT or INR normal unless DIC is there. Recognised classification for HELLP includes:

Tennessee system	Mississippi system
Abnormal peripheral smear for diagnosis of intramuscular hemolysis, s.bilirubin>1.2mg/dl LDH>600IU/L	AST>40IU/L and
AST>70IU/L	ClassI:platelet<50×10^9/L
LDH>600IU/L	ClassII:platelet 50-100×10^9/L
Platelets<100×10^9/L	ClassIII:platelet 100-150×10^9/L

Management of HELLP includes hospitalization for stabilization of hypertension ,DIC ,seizure prophylaxis and fetal monitoring.Definitive therapy is only delivery.Transfusion of blood or blood product for correction of anemia and coagulopathy are recommended.If platelets doesn't increase after 96 hrs after delivery ,it indicated severe condition with possible development of multiple organ failure[15].

Maternal complications may include DIC,abruptio placental,eclampsia,pulmonary edema,ARDS,acute renal failure.

Acute Fatty Liver of Pregnancy (AFLP) is a rare disorder most common in third trimester.It is inherent as autosomal recessive trait with an incidence of 1 in 7000 to 1 in 20,000 pregnancy. It is more in primiparous women of more than 30 years and women with multifetal pregnancy with male fetus.During pregnancy levels of free fatty acid increases physiologically and oxidation of these fatty acid by mitochondria provides energy required for fetal growth.Mutation of G1528C gene leads to deficiency of long chain 3 hydroxyacyl CoA dehydrogenase(LCHAD) enzyme which is required for the oxidation of fatty acid.Mother heterozygous for fatty acid oxidation disorder with an affected fetus(homozygous for fatty acid oxidation disorder)can develop AFLP. [16] Fetal fatty acid accumulate and enters the maternal circulation and leads to fat deposition in hepatocytes and impaired hepatic function in mother.

Typical symptoms include nausea,vomiting,epigastric pain, fatigue, and progressive jaundice develop over several days to weeks.Progression to moderate to severe hypoglcemia, coagulopathy,encephalopathy,acute pancreatitis, marked decrease in antiprothrombin III activity and rapidly leading to frank liver failure.About 50% of patient have signs of preeclampsia.DIC is occurs in 80-100% of patients with AFLP as compared to 21% of patients with HELLP syndtome[13].

Diagnosis of AFLP is made on clinical and laboratory findings Laboratory findings includes increased aminotransferase levels approaching to 1000IU/L,raised PT,hyperbilirubinemia <10mg/dl,raised creatinine levels,uric acid levels,neutrophilia,thrombocytopenia, hypoalbuminemia.On liver biopsy microvesicular steatosis is seen.The Swansea diagnostic includes:

Abdominal pain & vomiting	Ascites
Polydipsia/polyuria	Encephalopathy
High bilirubin (>14micromol/L)	High AST/ALT(>42IU/L)
Hypoglycaemia (<4mmol/L)	High ammonia (>47 micromol/L)
High uric acid (>340micromol/L)	Creatinine (>150micromol/L)
Leucocytosis(>11×10⁶/L) (PT>14sec)	Coagulopathy

Management of AFLP includes maximal supportive care and expeditious delivery irrespective of period of gestation.In rare cases,liver transplantation may be required.Maternal mortality rate has been reduced from 75-80% to less than 10% because of early diagnosis, prompt delivery and better critical care.In postpartum period liver function usually returns to normal within a week.Screening for defects in fatty acid oxidation should be done in mother and children born to mother with AFLP.

Liver diseases coincidental to pregnancy

Acute viral hepatitis

Viral hepatitis is the most common cause of jaundice in pregnancy.Hepatitis E may be more severe during pregnancy whereas pregnancy does not affect most of the viral hepatitis(A,B,C,D).Perinatal mortality is increased because of preterm delivery but viral hepatitis doesn't appear to be teratogenic.Symptoms may include nausea,vomiting,malaise,headache.Serum transaminases elevated ranging from 400-4000U/L,serum bilirubin levels are increased (5-20mg/dl), not associated with preeclampsia.

Hepatits A virus infection during pregnancy has similar prognosis as that in non pregnant patient and is self limited.

Hepatitis B is the most common cause of acute viral hepatitis in pregnancy.Presence of HBeAg is associated with high risk of neonatal infection.All pregnant women are screened for hepatitis B as a part of routine antenatal screening.The prevalence of neonatal infection depends on duration of gestation when maternal infection occurs, rare in first trimester and 67%in third trimester[9]. Infants born to HBsAg positive mother should receive single dose of HBIG and first dose of hepatitis B vaccine within 12hrs of birth and second dose at 1 month and third dose at 6 month of age.

Hepatitis C virus infection during pregnancy has similar presentation to non pregnant women.Risk of vertical transmission is low.Antiviral treatment during pregnancy is contraindicated.

Hepatitis D virus co-infect with hepatitis B virus.When present,incidence of acute hepatic failure increases.

Hepatitis E virus is associated with higher incidence of fulminant hepatic failure and mortality in pregnant women.

Other Causes of Jaundice in Pregnancy

- Leptospirosis, malaria, sickle cell crisis and hemolytic jaundice are treated as in nonpregnant woman
- Gall-stones: Pregnancy increases cholelithiasis. It may present as biliary colic, acute cholecystitis or acute pancreatitis. USG is helpful in diagnosis x ERCP with minimal fluoroscopy can be done
- Cholecystectomy may be required, open or laparoscopic depending up on stage of gestation.

Drugs: Drug induced cholestasis can present with asymptomatic disease where the only clinical manifestation is an elevation in alkaline phosphatase. Moreover, the target of injury can vary from a mixed hepatocellular cholestatic injury, to impairment of canalicular bile flow resulting in pure intrahepatic cholestasis, or to an "obstructive" drug induced cholangiopathy where the initial site of injury is located at various levels of the bile duct epithelium. Nitrofurantoin, anabolic steroids, chlorpromazine, prochlorperazine, cimetidine, erythromycin, estrogen, and statins can cause cholestasis and jaundice. Methyldopa and labetalol have been associated with hepatotoxicity including liver failure.

Liver tumors (adenoma) can enlarge and rupture during pregnancy so pregnancy is contraindicated in presence of unresected adenoma

Pregnancy in pre-existing liver disease

Chronic Hepatitis : Continuing hepatic necrosis, inflammation and fibrosis leading to cirrhosis due to chronic infection with HBV, HCV viruses or autoimmune chronic hepatitis Pregnancy outcome depends up on intensity of disease and presence of portal hypertension. Long term prognosis is poor so woman should be counseled regarding possibility of liver transplantation. Termination of pregnancy and sterilization.

Autoimmune Hepatitis : Autoimmune hepatitis is a progressive liver disease that predominantly affects women of all ages and can manifest at any time during gestation and the postpartum period. The disease activity is usually attenuated during pregnancy, and dosages of medication can be decreased because of the state of immune tolerance induced by the pregnancy. Flares occur in 11% of patients during gestation and up to 25% in the postpartum period. There is an increased risk of prematurity, low-birth-weight infants, and fetal loss. Pregnancy does not contraindicate immunosuppressive therapy. Both prednisone and azathioprine (FDA category D at dosages at dosages < 100 mg/d are considered safe during pregnancy and lactation

Conclusion

Liver diseases in pregnancy can lead to increased maternal and fetal morbidity and mortality.Extreme vigilance in recognizing clinical and laboratory abnormalities in pregnancy could lead to early intervention and successful outcome

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Understanding Epilepsy in Pregnancy

Dr Sarika Gautam
Professor, Deptt. of Obstetrics and Gynaecology
PGIMS, Rohtak
sarika.gautam07@gmail.com
Author

Dr Meenakshi B Chauhan
Senior Professor and Head of Unit III,
Deptt. of Obstetrics and Gynaecology, PGIMS, Rohtak
mbc51490@gmail.com
Co-Author

Introduction and Overview

Epilepsy is a neurological disorder characterised by recurrent, unprovoked seizures. It occurs because of aberrant electrical activity in the brain, which temporarily disrupts normal brain function. Seizures can take many forms, including convulsions, loss of consciousness, and altered awareness.[1]

Epilepsy is one of the most frequent neurological disorders during pregnancy, with a prevalence of 0.5–1%. Women of reproductive age account for almost one-third of all epilepsy cases. Pregnant women with epilepsy (WWE) are 10 times more likely to succumb than those who do not have the illness. [2]

Pregnancy can influence the treatment and progression of epilepsy, and epilepsy can influence pregnancy outcomes. Epilepsy therapy during pregnancy is critical for reducing hazards to both the mother and the developing foetus. Understanding the relationship between epilepsy and pregnancy allows healthcare professionals to give better treatment and support to pregnant women with epilepsy.

Sudden unexpected death in epilepsy (SUDEP) is defined as sudden, unexpected, witnessed, or unwitnessed death in epilepsy patients. Uncontrolled tonic-clonic seizures are the strongest risk factor for SUDEP in pregnant WWE. It excludes documented status epilepticus. A postmortem examination does not reveal the toxicologic or anatomic cause of death. There is an increased risk of mortality during the third trimester and postpartum period. A study done by MacDonald S et al. suggested that women with epilepsy are at increased risk of adverse outcomes, including death, during delivery hospitalisation, requiring urgent clinical attention. The risk of death increased by 10-fold compared to women without epilepsy. Women who have had seizures within the year before getting pregnant need to have their epilepsy closely monitored.

So, addressing the specific requirements during pregnancy and challenges that pregnant women with epilepsy encounter can help them to live a better life during this vital time.

Classification of epilepsy:

Classification of seizures is based on their onset; it could be focal, generalised, or unknown onset.[4]

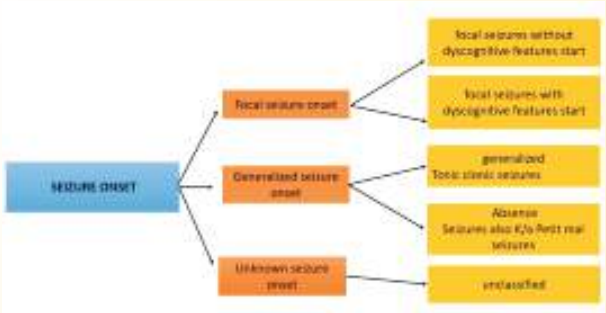

Fig 1. Classification of epilepsy according to onset of seizure activity:

Physiological Changes During Pregnancy:

Pregnancy triggers hormonal fluctuations, including oestrogen and progesterone levels, which can impact seizure threshold and frequency in epilepsy patients. Oestrogen may reduce seizure frequency, while progesterone may increase activity. Pregnancy can alter antiepileptic drug pharmacokinetics due to increased plasma volume and hepatic blood flow. This can lead to lower drug concentrations and decreased efficacy. Close monitoring is necessary for optimal seizure control and foetal risks.[5]

During pregnancy, factors like hormonal fluctuations, neurotransmitter levels, and cerebral blood flow can influence the seizure threshold, affecting the level of

stimulation needed to trigger a seizure.[6]

Some women's seizure thresholds may fall during pregnancy, increasing the likelihood of recurrence. Others may see a transient improvement in seizure control, especially during the second and third trimesters.

Understanding pregnancy physiological changes is crucial for healthcare providers to manage epilepsy effectively, optimise seizure control, and ensure the safety and well-being of both the mother and the developing foetus.

Congenital malformation and Antiepileptic Drugs:

The use of antiepileptic drugs (AEDs) during pregnancy is associated with a risk of congenital malformations in the developing fetus.

Risk Factors:

AEDs during pregnancy can cause structural abnormalities in the developing fetus, with the risk varying based on dosage, duration, and maternal health. First trimester exposure is the highest risk. Multiple AEDs concurrently increase congenital anomalies risk

Specific AEDs and Risks:

When discussing antiepileptic drugs (AEDs) and their potential to cause congenital malformations, it is important to consider the classification of these drugs based on their enzyme-inducing or non-enzyme-inducing properties.

Enzyme inducer AED	Non enzyme inducer AED
These drugs are having high risks of congenital malformation Phenytoin Carbamazepine Phenobarbital Primidone	These drugs are preferred during pregnancy due to their lower risk of drug interactions and favourable safety profiles for congenital malformations. Common Non-Enzyme-Inducing AEDs: Lamotrigine Levetiracetam Gabapentin Topiramate (some debate as it may have a mild inducing effect) Sodium valproate

Congenital Malformation associated with Commonly used AED's:

Phenytoin, Carbamazepine, Phenobarbital, lamotrigine, Sodium Valproate, Levetiracetam topiramate all these drugs are associated with risks of congenital malformations like Fetal Hydantoin syndrome, Cleft Lip and Palate, Cardiac Defects, Digital Hypoplasia, Growth Retardation, low IQ scores, theoretical risks of skeletal abnormalities, low IQ scores in offsprings. Choosing an appropriate AED is a tough decision and it requires judicious decision in lowest possible dose with single drug therapy.[4]

Antiepileptic Drug Selection and Considerations:

Selecting the appropriate antiepileptic drug (AED) for pregnant women with epilepsy involves careful consideration of several factors. The primary goal is to maintain optimal seizure control while minimizing risks to both the mother and the developing fetus. When choosing an AED, healthcare providers consider the woman's seizure type and frequency, previous response to medications, potential side effects, and the teratogenicity profile of the drug. AEDs like levetiracetam and lamotrigine are preferred due to lower birth defects risks. However, individualized treatment is crucial. Regular monitoring and adjustments are essential during pregnancy. Healthcare team discussions should consider benefits and risks, considering pregnancy stage and health. Collaboration between neurologists, obstetricians, and specialists is crucial for personalized treatment plans. Close monitoring is essential for optimal seizure control and minimizing risks.

Monotherapy vs Polytherapy:

Monotherapy should be preferred over polytherapy due to its lower risk of adverse effects on the developing fetus, reduced drug interactions, easier monitoring of medication levels, and lower risk of congenital malformations. The choice of medication depends on seizure type, response to previous medications, and potential risks. Regular prenatal visits and medication adjustments are crucial for maternal and fetal well-being.[7]

Following points should be considered while considering monotherapy:

- Preferable Option
- Benefits to avoid congenital malformation
- Seizure Control with lowest possible doses
- Medication Selection Factors: depends on type of seizures, previous medication response, fetal risks.
- Monitoring
- Adjustments During Pregnancy
- Dosage adjustments

Preconception Planning and Counselling for Women with Epilepsy:

Preconception counseling plays a pivotal role in achieving

optimal maternal health and minimizing risks to the developing fetus. It emphasizes a collaborative approach, involving neurologists, obstetricians, and healthcare providers. This proactive planning ensures tailored care, addressing epilepsy management, medication adjustments, and overall health to enhance pregnancy outcomes and maternal-fetal well-being. It must be kept in mind that at least six months before conception, seizure control should be attained. If clinically attainable, the lowest effective dose of a single anticonvulsant should be used, depending on the type of epilepsy.[8]

Following points to be included in preconception counseling:

Preconception Planning and Counseling for Women with Epilepsy	Points to consider for discussion
Timing of Conception	Discuss ideal conception time, AED adjustments.
AED Management	Review, cautiously adjust medications for pregnancy. Avoid sudden changes without medical advice.
Folic Acid Supplementation	Advice folic acid for neural tube defect prevention. Pre pregnancy folic acid 5 mg/day may be helpful in reducing the risk of AED-related cognitive deficits.
Medical Evaluation	Pre-pregnancy comprehensive health assessment.Monitor seizuresAED levelsoverall health
Risk Assessment and Counseling	Explain pregnancy risks, impact of epilepsy, and medications. Discuss personalized care plans.
Genetic Counseling	Discuss genetic risksgive counseling options for genetic epilepsy. Referral to genetic counsellor if needed.
Lifestyle Factors	Counsel on healthy habits, risks of alcohol along with pregnancy, smoking, drugs. Emphasize balanced dietregular exerciseSleeping pattern
Psychosocial Support	Address emotional needs and stress management. Offer support resources, counseling options for well-being.
Contraception and Family Planning	Explore safe contraception options, plan for future pregnancies. Discuss contraceptive management.

Risks and Complications Associated with Epilepsy in Pregnancy:

Epilepsy during pregnancy presents unique challenges due to hormonal changes and physiological adaptations. Managing epilepsy requires careful consideration of risks and complications, few risks and complications are as follows:[9]

i. Increased seizure frequency during pregnancy.
ii. Risk of status epilepticus.
iii. Potential risks of certain antiepileptic drugs (AEDs).
iv. Maternal injury during seizures.
v. Complications during labor and delivery.
vi. Higher likelihood of developing hypertensive disorders.
vii. Increased risk of premature birth.
viii. Babies may have lower birth weights and need specialized monitoring.
ix. Postpartum seizure risk after pregnancy or delivery complications.

Monitoring Fetal and Maternal Health Throughout Pregnancy:

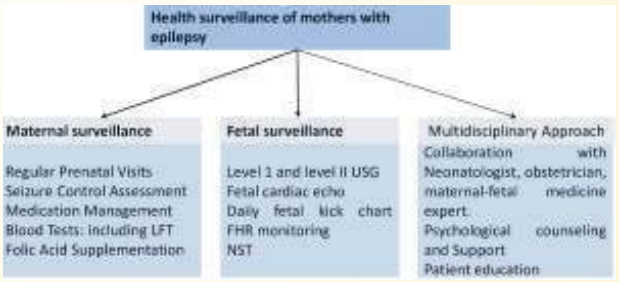

Fig 2: maternal health surveillance for Epilepsy in pregnancy

Management of Seizure Control during Pregnancy:

Regular intake of drug is emphasized in the antenatal period for optimal drug levels still there are chances of seizure episode during pregnancy.[2]

If a patient had fit following measures to be taken:

Action	Plan of action
Assessment	Quickly assess the situation and ensure the woman is in a safe environment. Check for any signs of injury or complications.
call for Assistance	If not already present, call for nursing staff or additional medical support. Inform them of the woman's history of epilepsy and the current situation. Divide the task among the team for emergency.
Protect the Airway	Ensure the woman's airway is clear and unobstructed. Place her in a side-lying position to prevent choking and allow fluids to drain. Use suction to clear the fluids. Try to avoid oral injuries by putting mouth gag if possible.
Monitor Vital Signs	Check the woman's vital signs, including pulse, blood pressure, and oxygen saturation.
Time the Seizure	Noting of time of onset and waning of seizure is important to decide type of seizure episode and if mother is having any other episode after first episode which may further change course of the action.
Avoid Restraints	Do not restrain the woman's movements during the seizure. Allow the seizure to run its course safely.
Protect Head injury	Place a soft object or cushion under the woman's head to prevent head injury. Ensure the area around her is clear of any sharp objects.
Medication Administration	Go stepwiseFirst line management is Benzodiazepines:IV lorazepam or IV diazepam are common first-line treatments.Administer a bolus of lorazepam 0.1 mg/kg i.e 5-10 mg but rate should be no more than 2mg/min.If seizure persists then consider loading dose of AED's.Preferred choice are levetiracetam give 1.0 gram loading IV slow followed by maintenance of 100 mg IV eight hourly which further can be switched to oral medication.If levetiracetam is not available consider phenytoin, load with 20mg/kg at a rate not more than 100mg/min.
Prepare for Possible Complications	Prepare mentally for management of aspiration, various injuries. Collaboration with intensivists, neurologist, ICU personnel.
Continuous Monitoring	Maternal and fetal monitoring to be done once the seizure settles.
Fetal monitoring and surveillance	Monitor fetal heart rate and patterns and do non stress test in third trimester.
Blood investigations	Blood sugar, serum electrolytes, blood gas analysis.
Postictal management	After the seizure, the woman may enter a postictal state characterized by confusion or drowsiness. Monitor her closely and provide reassurance.
Document the episode for future care	Record details of the seizure episode, including its duration, medications given, and response. This information is important for ongoing care and treatment planning.

Management of Epilepsy During Labour and Delivery:

Routine management of labour should be done. Diagnosis of epilepsy is not a basis for cesarean section or labor induction, and early delivery is not recommended for women with epilepsy unless obstetric risk factors are well controlled.

A caesarean section may be considered for a small percentage of women with significant increases in seizures and a high risk of status epilepticus. Pregnant women with epilepsy should be informed that the risk of seizures during labour and after delivery is low, approximately – 2%. Adequate pain management and care during labour are crucial to minimising risk factors. Epidural anaesthesia and nitrous oxide can reduce seizure risk, but pethidine is contraindicated due to lowered seizure thresholds. Epilepsy and pregnancy do not indicate surgical delivery, and the method of delivery is determined by obstetric conditions and neurological conditions.

If a patient had a seizure episode during labour, she should be managed according to the guidelines given for seizure management.

Management of Status Epilepticus:

Status epilepticus (SE) during pregnancy is a medical emergency that requires prompt and effective management to prevent potential harm to both the mother and the developing fetus. Status epilepticus is characterised by prolonged or repetitive seizures that last longer than 5 minutes without recovery of consciousness between seizures. Ensure the airway and administer

benzodiazepines like lorazepam. Second-line AEDs, such as phenytoin, may follow. Refractory cases need ICU care with EEG monitoring. Monitor both the mother's and the foetus's condition. Post-episode, continue care with neurology assessments and pregnancy monitoring.

Postpartum Care for WWE:

Postpartum care for women with epilepsy involves monitoring for heightened seizure risk due to hormonal changes and stress. Continued AED management, adjusted as needed, is crucial. Seizure activity should be closely monitored post-delivery, with any changes reported promptly. Physical recovery, medication compatibility with breastfeeding, and emotional support, including awareness of postpartum depression risks, are key aspects of comprehensive postpartum care.

Contraception and Family Planning:

Contraceptive Choices:

Discuss contraceptive options with healthcare providers to choose a method that is safe and effective. Some AEDs may interact with hormonal contraceptives, so careful consideration is necessary. Copper IUDs, LNG-IUS, and medroxyprogesterone acetate injections should be promoted as effective contraceptive techniques that are not impacted by enzyme-inducing AEDs. Women taking enzyme-inducing AEDs should be informed about the risk of failure with hormonal contraceptives like OCPs, vaginal rings, and patches, as their efficacy may be affected. Women taking non-enzyme-inducing AEDs like sodium valproate, levetiracetam, gabapentin, vigabatrin, tiagabine, and pregabalin can benefit from having access to all contraception options.

Family Planning Discussions:

Women with epilepsy should have discussions about family planning for future pregnancies. Preconception counselling can help address potential risks and optimise outcomes for subsequent pregnancies.

Long-Term Health Monitoring:

Regular Follow-ups:

Continued follow-up appointments with neurologists and obstetricians are essential for long-term health monitoring. This includes monitoring for any potential side effects of AEDs and assessing overall health.

Education and Awareness:

Ongoing education about epilepsy management, including triggers and warning signs of seizures, is important. Awareness of potential risks and proactive measures can help women manage their condition effectively.

In conclusion, managing epilepsy during pregnancy requires a multifaceted approach involving careful preconception planning, medication adjustments, and continuous monitoring of both maternal and foetal well-being. Prompt recognition and treatment of seizures, along with collaboration between neurologists and obstetricians, are essential. Postpartum care, including monitoring for heightened seizure risk and providing psychological support, ensures the best outcomes for both mother and baby in this delicate journey of epilepsy and pregnancy.

Key Points:

- The diagnosis of epilepsy should be made by a subject expert who is a neurologist.
- Women with epilepsy, their families, and health care providers should be aware of the type of epilepsy to assess its risk.
- Women with epilepsy should receive preconceptional care to assess their risks for inheritance.
- Seizures should be considered an emergency and treated accordingly.
- Antiepileptic drugs should be chosen wisely with a minimum effective dose.
- Routine monitoring of drug levels is not recommended.
- Regular antenatal visits are recommended, as per protocol.
- Sonography to rule out congenital malformation at 18 to 20 weeks of gestation.
- A foetal cardiac echo should be done to rule out cardiac defects.
- Postpartum counselling and advice on contraception should be done.

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"It is not until you become a mother that your judgment slowly turns to compassion and understanding."

–Erma Bombeck

"All women become like their mothers.
That is their tragedy.
No man does. That's his."

–Oscar Wilde

Endocrine Emergencies In Obstetrics

Dr Amanjot Kaur
Assistant Professor
Department of Obstetrics & Gynaecology
Postgraduate Institute of Medical Education
and Research, Chandigarh, India
e-mail: aman50055@yahoo.com
First author

Dr Minakshi Rohilla
Professor
Department of Obstetrics & Gynaecology
Postgraduate Institute of Medical Education
and Research, Chandigarh, India
e-mail: minurohilla@yahoo.com
Second and corresponding author

Endocrinologic emergencies though rare, are associated with a high case fatality rate of 10-30%.[1] Pregnancy causes marked physiological changes and endocrinologic alterations. Not only are the classic clinical signs and laboratory parameters modified during pregnancy, the therapeutic options are also limited in order to prevent fetal insults. Prompt recognition and appropriate and timely intervention helps to salvage outcomes for both the expectant mother and the fetus. Diabetes and thyroid disorders are more common in the population and thus emergencies related to these disorders are more common.[2] The present chapter discusses some life-threatening endocrine emergencies presenting in pregnancy.

Diabetes Related Complications

DIABETIC KETOACIDOSIS (DKA)

DKA has an incidence of 2-3% and perinatal mortality rate of 10-20% in pregnancy.[3] The condition was undeniably fatal prior to discovery of insulin. It may lead to premature birth both as a result of premature labor, as well as from medical intervention.

Pregnancy increases the risk of ketoacidosis as there is a relative insulin deficiency in a state of insulin resistance in pregnancy. This is caused by increased the level of human placental growth hormone and human placental lactogen. Compensatory metabolic acidosis results from a combination of insulin deficit and decreased buffering capacity due to respiratory alkalosis of pregnancy.[4,5] The pathophysiology of DKA involves a lack of insulin causing a perceived hypoglycaemia in tissues of liver, fat and muscle. This results in release of glucagon stores causing hyperglycaemia, leading to osmotic diuresis, electrolyte depletion and hypovolemia. Counter regulatory hormones from the adipose tissue cause fatty acid release into the circulation. These fatty acids are metabolised to ketone bodies leading to metabolic acidosis which is evident as an anion gap on blood gas analysis. Ketoacids bind to sodium and potassium and are excreted in the urine leading to dyselectrolytemia. Uninhibited course of DKA leads to decrease in tissue perfusion causing renal dysfunction further leading to shock and its consequences and cardiac dysfunction due to dyselectrolytemia.[6]

Pregnancy is associated with an increased glucose requirement which leads to a switch from carbohydrate to fat metabolism This fatty acid metabolism leads to starvation ketosis in the mother which contributes to DKA. Thus acidosis in pregnant diabetics may present with DKA with normal blood glucose levels rather than hyperglycemia.[7]

Precipitating factors for DKA include protracted vomiting, infections, hyperemesis, and insulin non-compliance. Patients present with vomiting, nausea , abdominal pain, polyuria, polydipsia ,muscle weakness, fruity breath, altered mentation, hyperventilation (Kussmaul breathing), tachycardia, tachypnoea, shock and coma. These may be associated with foetal heart rate abnormalities like reduced variability, and variable , or late decelerations. Laboratory findings reveal acidosis, elevated plasma glucose(>250 mg/dl), elevated anion gap(>12mEq/L), hyperkalaemia, hyponatremia, base deficit of 4mEq/L or greater, raised urinary and serum ketones and elevated renal function tests secondary to dehydration. Aggressive fluid administration, intravenous insulin , and correction of electrolyte disturbance (Table 1) form the pillars of treatment of DKA Management should be done in intensive care settings and entails continuous maternal and foetal monitoring.

DIABETIC KETOACIDOSIS : MANAGEMENT DURING PREGNANCY

INTRAVENUOUS FLUID THERAPY

1. i/v normal saline, 1-2litres/hour in first hour followed by 250 -500 ml/hour. If the corrected serum Na^+ levels are elevated, change to 0.45% NaCl
2. Maintain hourly charts for fluids, electrolytes, and laboratory parameters.
3. Start 5% dextrose with normal saline or 0.45% normal saline at 150 - 250 mL/h when plasma glucose reaches 200 mg/dL
4. After 8 to 24 hours switch to 5% dextrose with 0.45% NaCl at 125 mL/h.

INSULIN THERAPY

1. Give loading dose of regular insulin 0.1 to 0.2 U/kg as intravenous bolus dose as per plasma glucose levels.\
2. Start continuous insulin infusion at 0.1 U/kg/h.
3. Double the insulin infusion every hour if plasma glucose levels do not fall by 50 mg/dL in the first hour, till a steady glucose decrease is attained.
4. Reduce the infusion rate to 0.05-0.1 U/Kg/h when plasma glucose reaches 200mg/dL\
5. Maintain serum glucose 100-150 mg/dL until resolution of ketoacidosis.

POTASSIUM

1. Serum potassium < 3.3 mEq/L - stop insulin and administer potassium 20 -30 mEq/h till it is > 3.3 mEq/L after confirming adequate renal function.
2. Serum potassium - 3.3 - 5.3 mEq/L - add 20-30 mEq potassium per litre IV fluid to maintain the values at 4 - 5 mEq/L.3. Check serum potassium values every 2 hourly and maintain < 5.3 mEq/L.

FETAL MONITORING

1. Keep high threshold for delivery in case of non reassuring fetal heart status as an emergency caesarean delivery during a critical illness may further deteriorate the maternal status and lead to delivery of an acidotic fetus.
2. Fetal heart tracings take 4-8 hours to resolve in such circumstances.[8]

HYPOGLYCEMIA

Hypoglycemia is defined as glucose blood levels below 60mg/dL and may be life-threatening due to resultant convulsions or hypoglycemic coma. Patients under strict glucose levels' monitoring may develop the condition. Symptoms due to hypoglycemia like sweating and tremor are easily reversible by giving glucose. In the event of inability to swallow, glucagon should be administered. An effort should be made to rapidly identify and cooorect the cause of hypoglycemia e.g. correction of diet or insulin dosage. Psychiatric cases of pregnant women taking higher than required doses of insulin may be diagnosed by C-peptide levels.

THYROID STORM

Thyroid storm affects 1-2% of pregnant women suffering from hyperthyroidism. It is marked by a severe hypermetabolic state caused due to excessive endogenous thyroid hormones and has a case fatality rate of 10 -30%.[9] Patients presenting with thyroid storm have an underlying diagnosis of hyperthyroidism resulting from Graves disease, multi- nodular goitres, toxic adenomas, or hydatidiform moles. Thyroid storm occurs due to poor disease control and some precipitating factor like preeclampsia, infection, surgery, trauma, and in sometimes, even labour or cesarean delivery.[10] Pregnancy is associated with a higher risk for cardiac failure, and this risk gets elevated by 10% in thyroid storm.

A thorough search for underlying aetiology should be made. A high index of clinical suspicion is warranted as the presentation may be non-specific and may be confused with pregnancy associated conditions. Headache, raised blood pressure, pulmonary edema , cardiac failure which can be features of thyroid storm can all be present in the preeclampsia spectrum.

SIGNS AND SYMPTOMS :

Patient presents with symptoms of hypermetabolism : it includes hyperpyrexia, tachycardia, arrhythmias, and neurologic symptoms like altered mental status, restlessness and seizures, gastrointestinal symptoms like nausea, vomiting , hepatic dysfunction and sometimes congestive cardiac failure.. The clinical Burch-Wartofsky Point Scale scoring system[11] can be used for confirmation of clinical diagnosis and to grade the severity. This scale gives objective points for thermoregulatory dysfunction,

CNS Effects, gastrointestinal & hepatic dysfunction, cardiovascular dysfunction ,congestive cardiac failure, atrial fibrillation and precipitant history. A score of 45 & above indicates thyroid storm, 25–44 indicates impending throid storm and if less than 25, thyroid storm is unlikely.

On laboratory analysis, free T4 and free T3 are raised well above the normal limits of pregnancy. The Thyroid-stimulating hormone levels are generally very low or undetectable but should be interpreted with caution due to the effect of the human chorionic gonadotropin on thyroid gland. Additionally, there can be associated leucocytosis, transaminitis, hyperglycemia, hypocalcaemia, and dyselectrolytemia on metabolic screening.

MANAGEMENT

Patient should be managed in intensive care settings with multidisciplinary approach. Initial therapy includes supportive management with fluids, oxygen, management of hyperpyrexia and correction of dyselectrolytemia. Acetaminophen is the antipyretic of choice and is preferred over aspirin as salicylates displace FT4 from the binding proteins thus increase its circulating levels.[12]

Anti Thyroid Drugs(ATDs) form the mainstay of treatment. Methamizole (MMI) is prescribed as 20 mg 6 hourly (80 mg total daily dose) . Propylthiouracil (PTU) is given as a loading dose of 400 mg followed by 200 mg 4 to 6 hourly (total of 800–1200 mg/d). PTU additionally inhibits the peripheral conversion of T4 to T3.[12] Iodine medications should be administered one hour after the thioamides to further block hormonal release. This one hour gap is essential to prevent the use of iodine as a substrate for TH synthesis which can worsen thyrotoxicosis. Iodine can be given as Lugol's solution, 10 drops thrice a day or potassium iodide 5 drops 6 hourly orally.

Beta blockers like propranolol are used to manage hyperadrenergic symptoms and also to decrease conversion of T4 to T3 at higher doses. Short acting beta blockers should be used in patients with cardiac dysfunction. High dose steroids therapy can be used to reduce conversion of T4 to T3in the periphery, to decrease relative adrenal insufficiency and to increase vasomotor stability. These steroids can be continued for 2-3 days with a careful monitoring of blood sugars. Hydrocortisone (50–100 mg 8 hourly) is favoured over dexamethasone (2–4 mg 8 hourly) as the latter may cross the placenta.

Digoxin may be needed in patients developing congestive cardiac failure. Bile acid sequestrants (cholestyramine 4 g 6–8 hourly) may help to decrease enterohepatic recycling of Thyroid Hormones and should be considered in severe cases. Early delivery should be avoided in as the fetal status may improve once the mother stabilizes. It takes 24 to 48 hours for clinical improvement to commense. In case of failure of same, dialysis or plasma exchange may be undertaken to decrease thyroid-stimulating hormone receptor antibodies (TRAb) and TH concentration. In the rare cases not responding to medical therapy, surgical treatment with definitive thyroidectomy may be done. Surgery is associated with significant risks during pregnancy, and should be undertaken by experienced surgeons. During follow up of patients after discharge, FT4 levels should be maintained at the upper limit of normal for the gestational age.

Hyperthyroidism and Adverse Perinatal Outcomes

Untreated overt hyperthyroidism is associated with increased risk of miscarriage (25%), preeclampsia(5 times) ,placental abruption, stillbirth (5-6%), premature delivery(15%) , foetal growth restriction, foetal/neonatal thyrotoxicosis, and new-born central hypothyroidism. Graves antibodies raised thrice the normal values place the foetus at risk for development of Graves' disease. In such circumstances, serial scans 4 weekly should be done to evaluate the foetal thyroid gland, heart rate, amniotic fluid index, foetal biometry ,bone maturation, evidence of hydrops and foetal cardiac dysfunction to delineate foetal risk. Hyperthyroidism in pregnancy should be managed in consultation with a maternal–foetal medicine specialist.

MYXEDEMA COMA

Myxoedema coma is characterized by severe hypothyroidism with multiorgan involvement and is very rare in pregnancy. It is difficult to diagnose, and suspicion should be raised by a prior history of hypothyroidism. Untreated or inadequately treated hypothyroidism may lead to myxoedema coma.

Precipitating factors may be infections, labour, congestive cardiac failure, cerebrovascular events, drugs like anaesthetic agents, antidepressants, neuroleptics, cold or inadequate treatment of hypothyroidism. The various signs and symptoms are hypothermia, coma, dyspnoea, generalized oedema, macroglossia, bradycardia, low volume pulse, blunted reflexes, constipation, thin and dry hair and seizures which can be both focal or generalized. Laboratory evaluation are generally suggestive of serum free T3 and T4 concentrations reduced and serum TSH values extremely raised than the normal reference range. The TSH values may exceed 100mIU/L in severe cases. There may be associated anemia, hyponatremia, hypoglycemia, raised transaminases , deranged renal functions, high serum creatine kinase and lactate dehydrogenase. There can be

associated hypoxemia, hypercapnia, and respiratory acidosis.[13]

Treatment protocol consists of levothyroxine along with hydrocortisone and supportive management. Intravenous levothyroxine 200 - 400 mcg is administered, followed by daily dose of 50 - 100 microgram, and triiodothyronine 5 - 20 microgram intravenously, followed by 2.5 -10 microgram eight hourly. Oral trearment can be commences once the patient starts tolerating orally. (an appropriate oral dose is i/v dose divided by 0.75).

Supportive measures include passive rewarming, appropriate intravenous fluids and vasopressors, antibiotics as indicated, continuous monitoring and mechanical ventilation if required.

HYPERTENSIVE CRISIS IN ENDOCRINE HYPERTENSION

Endocrine hypertension may result from pheochromocytomas which are catecholamine secreting tumors of adrenal medulla or extra-adrenal chromaffin tissue, and have an incidence of 0.002% of all pregnancies. The classical triad of symptoms consist of headache, tachycardia and sweating, though the symptoms may be variable. Patients may have paroxysmal or continuosly increased blood pressure leading to hypertensive crisis. Fetomaternal mortality is around 50% in undiagnosed and 4-11% in treated cases. Differentiating endocrine hypertension from from pregnancy induced conditions is challenging.[14] Plasma or urinary metanephrines help to clinch the diagnosis. An ultrasound of the abdomen has a sensitivity of 75–95% and MRI has a sensitivity of 98–100% for diagnosis in preganancy.. MIBG scintigraphy or FDG-PET can be done after delivery. Multidisciplinary management involving endocrinologists, obstetricians, and endocrine surgeons is important. For acute management of hypertension, urapidil is the drug of first choice. Nifedipine is considered safe after the first trimester and may be used alternatively. Methyldopa is contraindicated in pheochromocytoma patients. When diagnosed before 24 weeks, adrenalectomy should be performed by minimally invasive means. If diagnosed later, medically management should be done till delivery and caesarean section is the preferred mode for the same.

ADRENAL INSUFFICIENCY (AI) AND ADRENAL CRISIS

Adrenal insufficiency (AI) in pregnancy is generally autoimmune and predisposes to preterm birth and caesarean section. Pregnancy is generally associated with increased steroidogenesis , hence patients with known AI on glucocorticoid therapy may require to hike the dose, specially in third trimester of pregnancy. Hydrocortisone has transplacental passage, and may cause fetal adrenal cortical suppression which must be prevented. Neonate should be evaluated for AI. Acute adrenal crisis (''Addisonian Crisis'') may be precipitated in patients with known AI in conditions of physical stress like surgery, trauma, pain , delivery and infection. It may become difficult to diagnose this condition in pregnancy due to non-specific symptoms fatigue, vomiting, abdominal pain, adynamy and weight loss. Urgent and timely treatment of adrenal crisis is of utmost importance and consists of intravenuos hydrocortisone 100 mg followed by continuous infusion of the same at 100 mg/24 h. Additionally, adequate volume substitution is of vital importance.[15]

HYPOPHYSITIS

Lymphocytic hypophysitis is a rare autoimmune disease. Most(50%) patients present in the last month of pregnancy and two moths after delivery. It typically presents in fertile women and its association with pregnancy is incompletely understood. Patients present with signs of hypopituitarism, central diabetes insipidus, increased body mass, or cerebral mass effects like headache , visual field defects and hyperprolactinemia. The clinical spectrum can be variable with no symptoms at all to life threatening conditions. Spontaneous remissions may occur during pregnancy. Treatment depends on clinical symtomatology and primarily consists of replacement of pituitary hormones which are deficient in association with desmopressin administration. Glucocorticoids in high doses may be required. Severe cases like visual impairment may need Transsphenoidal surgery. Radiotherapy is contraindicated.[16]

KEY POINTS

1. Though endocrine emergencies are rare, they are associated with a high case fatality rate.
2. Diagnosis is difficult as many symptoms may be masked by pregnancy or may be similar to pregnancy symptoms.
3. Therapeutic options in pregnancy are limited to avoid fetal insult.
4. Multidisciplinary involvement and intensive care settings are required for management.
5. Diabetic ketoacidois and thyroid storm are the most common endocrinologic emergencies encountered in pregnancy.

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"A mother's love
for her child is like
nothing else in the world.
It knows no law, no pity,
it dares all things and
crushes down remorselessly
all that stands in its path."

–Agatha Christie

Congenital Heart Disease in Pregnancy

Dr. Amrita S Jaipuriar
Consultant Gynaecologist & Obstetrician
Jaipuriar Medical & Research Centre & Garg Hospital, Gorakhpur (U.P)

Introduction :

The prevalence of heart disease during pregnancy ranges from 0.3% to 3.5%. However, in the presence of maternal heart disease, the circulatory changes that occur during pregnancy can lead to the decompensation or death of the mother or fetus[1].

While most heart conditions can be managed well during pregnancy with positive outcomes, some cardiac conditions can lead to severe health problems for both the mother and baby.

The incidence and characteristics of heart disease during pregnancy differ significantly between high-income, industrialized countries (HIC) and low- and middle-income countries (LMIC). In Western countries, congenital heart disease (CHD) is the most prevalent type of heart disease during pregnancy. In contrast, in LMIC, rheumatic heart disease (RHD) accounts for up to 88% to 90% of cases. However, as healthcare coverage has increased, the incidence of RHD has decreased, and the ratio of RHD to CHD has decreased from 10:1 to 3:1 in developing countries[2].

Simple Congenital Heart Disease lesions includes mild Pulmonary valve stenosis, uncompleated ASD, VSD or PDA and complex Congenital Heart Diseases include complicated Anatomical and Physiological lesions .Still simple lesions as Atrial Septal defect can be complicated with Pulmonary hypertension or Atrial fibrillation and this will need higher level of care.

Thus the categorisation of patients is very helpful in the Management.

Physiological change in pregnancy :

ANTEPARTUM

(A) Maternal Blood volume increases by 40% for a singleton and 67% for twins. Peak values are there at 32 week of gestation.

Plasma volume increases by 45% to 55% and Red cell mass by 20% to 30%.

This causes Physiological anaemia of pregnancy.

Estrogen plays the key role in plasma volume expansion and promotes sodium and water retention by upregulating the production of Angiotensin, Renin and Aldosterone.

(B) The median level of B type natriuretic peptide in normal pregnant women is twice of non pregnant. It remains high throughout the pregnancy untill 72 hours after delevery. Maternal outcomes are poor if BNP is more than 100pg/ml.

(C) Circulating Albumin Consentration fall 12% to 18% in pregnancy with lowest level at 24 weeks of gestation. Decreased oncotic pressure and uterine compression of Inferior Vena Cava causes oedema of pregnancy.

(D) Maternal cardiac output increases by 30% to 50% in singleton pregnancy with an additional 10% to 20% increment in a twin pregnancy

(E) Maternal heart rate rises 10 to 20 beats/ minute over pre pregnancy rate and reaches a peak in the late second or early third trimester.

(F) Arterial Vasculature – Vascular tree undergoes remodeling to accommodate increased blood voulme.

These structrual alterations increase the risk of Aortic route enlargement and dissection in pregnant patients with Marfan's syndrome or other aortopathies.

Pulmonary vascular resistance declines by 24% which accomodates the increase in pulmonary flow.

There is also a fall in systemic vascular resistance and it is minimum at 24 weeks pregnancy.

G) Tidal volume increases by 40%. There is also a proportional increase in Minute ventilation. This physiological hyperventilation is greater than the increase in oxygen consumption and accounts for the breathlesness in early pregnancy.

INTRAPARTUM

Labour increases the heart rate, central venous pressure an cardiac output. Systemic arterial pressure rises with progration of labour.

Uterine contractions augument maternal cardiac activity because of anxiety and pain, also expulsion of uterine blood flow into central venous circulation leads to increase in cardiac output. Overall maternal cardiac output increases 10% to 30% during first stage and 50% in the second stage.

Effective pain control and lateral tilt in labour can help to keep the BP attenuated.

Valsalva maneuver elicits a moderate transient fall in cardiac output resulting from decreased venous return. So, Forceps or Vacuum extraction should be preferred in patients with Aortic stenosis, Pulonary hypertension and Aortopathies.

POSTPARTUM

Post delivery there is increased preload via Venacaval decompression and extrusion of blood from the contracted uterus into the Inferior VenaCava.

This leads to increase in cardiac output by 60% to 80%. Mean artrial pressure (MAP) can be elevated for one to two days Postpartum.

Table 1. Haemodynamic changes in pregnancy.[5-7]

Haemodynamic parameter	Pregnancy	Clinical implication	Normal findings
Blood flow	↑	• Nose bleeds common • Baseline serum creatinine lower in pregnancy	• Bounding/collapsing pulse • Prominent non-displaced apical pulse
Blood volume (plasma and RBC)	↑	• Physiological anaemia in pregnancy • Higher risk of cardiac failure in multiple pregnancy	• Ejection systolic murmur • Loud first heart sound • Third heart sound
Systemic vascular resistance	↓	• Risk of maternal fetal compromise in women with fixed cardiac outputs (stenotic lesions)	• Venous hum • Mammary souffle
Stroke volume	↑		• Relative sinus tachycardia (10–20 bpm)
Cardiac output	↑	• Sinus tachycardia towards end of pregnancy	• Ectopic beats
Heart rate	↑		• Peripheral oedema
Blood pressure	↓		• Warm/erythematous extremities
Pulmonary capillary wedge pressure	↔	• Increased susceptibility to pulmonary oedema	• Elevated JVP in late pregnancy
Colloid oncotic pressure	↓		
Central venous pressure	↔		
Maternal oxygen consumption	↑	• Tendency to ischaemia in pregnant women with cardiac disease	

bpm = beats per minute; RBC = red blood cell; JVP = jugular venous pressure.

Counseling, Assesment and Evaluation

Potential adverse outcomes in the mother with Congenital Heart Disease include stroke, arrhythmia, pulmonary oedema and death. For the fetus, growth restriction and fetal loss are more common.

Any uncorrected or inadequately corrected congenital heart disease that is associated with cyanosis is associated with an increased risk of miscarriage, poor fetal growth, prematurity and a small-for gestation fetus, especially in women with resting arterial saturation <85% or haemoglobin >18 g/dl and haemato crit >55%.

Asymptomatic women with simple defects or previously repaired defects tolerate pregnancy well.

Women with congenital heart disease are at increased risk of having babies with congenital heart defects and so should be offered specialist fetal cardiac scans between 18 and 20 weeks.

Preconception counseling is important. Impact of pregnancy on heart disease should be discussed.

Cardiovascular risk assesment should be done and effect on the offspring should be informed.

Evaluation of maternal risk and treatment or repair of the defects should be advised and done prior to conception.

Cadiac status assesment is mandatory beginning from Arterial Saturation, ECG, Echocardiography, MRI and exercise test if necessary. Other co-morbodities should be looked for and ruled out.

Delivary plan and Anaethesia should be discussed.

- Advancements in the medical and surgical care of CHD patients have improved survival rates to adulthood. CHDs comprise a broad spectrum of anatomic abnormalities but have limited pathophysiologic states. Optimum care for these patients requires a coordinated effort by a cardio-obstetric team, as they need careful antenatal and postnatal surveillance and an individualized plan for labor and delivery.
- Pregnant women with CHD would require appropriate preconception assessment, risk stratification, and counseling. A cardio-obstetric team can prevent severe complications by identifying high-risk patients and providing timely, proper management. It is essential to bridge the gap between HIC and LMIC countries by implementing careful antenatal and postnatal surveillance and developing individualized plans for labor and delivery[3].
- Cardiac complications during pregnancy, including RHD, CHD, ischemic heart conditions, and cardiomyopathies, present unique challenges due to physiological changes and increased stress on the maternal cardiovascular system.

Guidelines:

- A team of cardiovascular and obstetrics experts should evaluate the patient's health condition before conception, optimize her cardiac status if required, and provide support throughout pregnancy and postpartum. The modified WHO classification is primarily used to assess the risk of certain conditions

before pregnancy (Table 1). However, women with modified World Health Organization class III and IV conditions require an experienced cardio-obstetrics team to manage complex cardiac disease during pregnancy[4, 5].

Table 2 - Modified WHO (m WHO) risk stratification classification, Pregnancy care, and delivery location for women with pre-existing cardiovascular disease[4,5, 7].

NYHA Classification – (Table-3)

The Stages of Heart Failure:

1- Class I - No symptoms and no limitation in ordinary physical activity, e.g. shortness of breath when walking, climbing stairs etc.

2- Class II - Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.

3- Class III - Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20—100 m).Comfortable only at rest.

4- Class IV - Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients.

5- No NYHA class listed or unable to determine.

Table 4 –Classes of Recommendation[6](ESC 2018 Guidelines for Cardiovascular disease during pregnancy)

Classes of Recommendations	Definition	Suggested wording to use
Class I	Evidence and general agreement that a given treatment or procedure is beneficial, practical, effective	Is recommended/ indicated
Class II	Conflicting evidence and a divergence of opinion about the usefulness / efficacy of the given treatment or procedure	
Class II a	The weight of evidence /opinion is in favor of usefulness /efficacy	Should be considered
Class II b	Usefulness/efficacy is less well established by evidence /opinion	May be considered
Class III	There is evidence or general agreement that the given treatment or procedure is not useful/effective and, in some cases, may be harmful.	Is not recommended

The 2018 ESC guidelines for congenital heart disease in pregnancy offer evidence-based recommendations for managing cardiovascular diseases during pregnancy. They provide a framework for pre-pregnancy counseling, risk assessment, and management strategies to ensure the safety of both the mother and the developing fetus.

Table 5 – ESC 2018 Recommendations for Congenital Heart Disease[6]

Recommendations	Class	Levels
Patients with a systemic right ventricle (Mustard/benign or congenitally corrected TGA) in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR should be advised against pregnancy.	IIa	C
Anticoagulation treatment should be considered during pregnancy in Fontan patients.	IIa	C
Symptomatic patients with Ebstein's anomaly with saturations <85% and heart failure should be advised against pregnancy.	IIa	C
In patients with a Fontan circulation and saturations <85%, depressed ventricular function, moderate–severe AV regurgitation, refractory arrhythmia, or protein-losing enteropathy, pregnancy is not recommended.	III	C

Table 6

Investigation	Interpretations
Natriuretic Peptides	BNP levels >100 pg/mL and NT-proBNP>450 pg/mL suggest heart failure in non-pregnant patients.Serial BNP-level measurements during pregnancy and postpartum can aid in clinical decision-making.BNP levels can determine cardiac decompensation in pregnancy, with elevated levels indicating potential complications from the second trimester.
Cardiac Troponin I, Troponin T, and"High-Sensitivity" Troponin	Pregnant and postpartum patients with chest pain should get a troponin test and ECG to check for acute coronary syndrome.Troponin I levels may be slightly elevated in postpartum women with severe preeclampsia and other non-cardiac conditions. Cardiology consultation should be sought as needed.
Electrocardiogram (ECG)	An ECGshould be performed in pregnant women presenting with chest pain, shortness of breath, or palpitations to assess for features of ischemia, infarction, or arrhythmias.ST-wave and T-wave abnormalities are common during pregnancy, usually in the left precordial leads, and tend to resolve after delivery.Further assessment is recommended if any irregularities in the rhythm are observed on the electrocardiogram (ECG).
Echocardiogram	Pregnant or postpartum women with heart disease, valvular and aortic disease, cardiomyopathies, and those exposed to cardiotoxic chemotherapy should undergo an echocardiogram.Women with certain conditions like pulmonary hypertension or unexplained oxygen desaturation should have an echocardiogram before and during pregnancy.
Exercise Stress Test	Exercise stress test is recommended for heart patients planning pregnancy. The submaximal tests are suggested for asymptomatic pregnant patients with suspected heart disease, as per international guidelines
Computed Tomography (CT Scan)	Perform CT scan in pregnant or postpartum women with suspected pulmonary embolism or acute aortic dissection.Use contrast agents only when necessary for crucial diagnostic information affecting care.
Magnetic Resonance Imaging (MRI)	MRI is rarely used for urgent cardiovascular evaluation during pregnancy due to limited availability and time-consuming nature compared to CT.MRI is the preferred imaging modality for pregnant women to assess heart function when echocardiography is non-diagnostic.Limit the use of gadolinium in pregnant patients for MRI scans. Only use it if it improves diagnostic performance and is expected to improve fetal or maternal outcomes.
Holter Monitor or Prolonged Cardiac Monitoring Device	A Holter monitor or a wireless patch cardiac monitor can help assess palpitations, lightheadedness, and syncope during pregnancy.

Recommendations and Conclusions ACOG Practice Bulletin -

Avoid pregnancy or consider abortion if the patienthas severe heart disease (ejection fraction less than 30%, class III/IV heart failure, severe valvular stenosis, Marfan syndrome with aortic diameter >45mm, bicuspid aortic valve with aortic diameter >50mm, or pulmonary arterial hypertension).

CHD in pregnant women should prompt screening for fetal echocardiography, and vice versa; detection of CHD in a fetus or neonate may prompt screening for parental CHD.

For women with complex congenital or non-congenital heart disease, treatment by a Pregnancy Heart Team is recommended.

Table -7

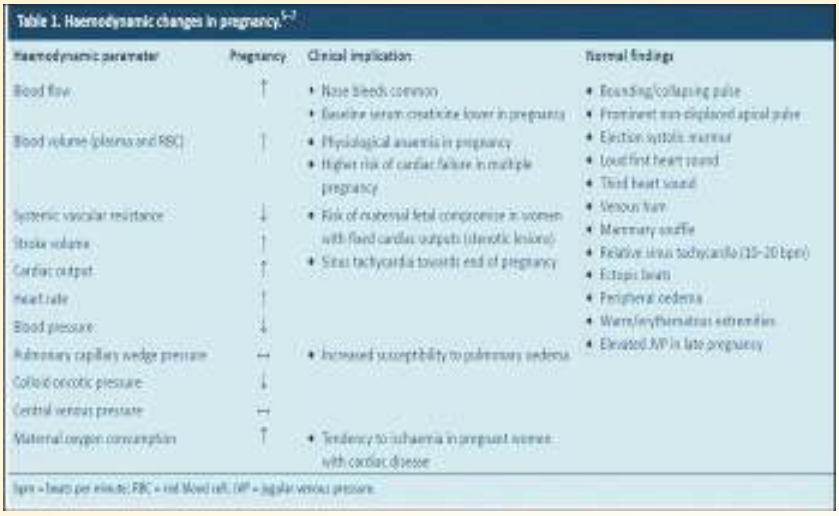


Table 1. Haemodynamic changes in pregnancy.[5–7]

Haemodynamic parameter	Pregnancy	Clinical implication	Normal findings
Blood flow	↑	• Nose bleeds common • Baseline serum creatinine lower in pregnancy	• Bounding/collapsing pulse • Prominent non-displaced apical pulse
Blood volume (plasma and RBC)	↑	• Physiological anaemia in pregnancy • Higher risk of cardiac failure in multiple pregnancy	• Ejection systolic murmur • Loud first heart sound • Third heart sound
Systemic vascular resistance	↓	• Risk of maternal-fetal compromise in women with fixed cardiac outputs (stenotic lesions)	• Venous hum • Mammary souffle
Stroke volume	↑	• Sinus tachycardia towards end of pregnancy	• Relative sinus tachycardia (15–20 bpm)
Cardiac output	↑		• Ectopic beats
Heart rate	↑		• Peripheral oedema
Blood pressure	↓		• Warm/erythematous extremities
Pulmonary capillary wedge pressure	↔	• Increased susceptibility to pulmonary oedema	• Elevated JVP in late pregnancy
Colloid oncotic pressure	↓		
Central venous pressure	↔		
Maternal oxygen consumption	↑	• Tendency to ischaemia in pregnant women with cardiac disease	

bpm = beats per minute; RBC = red blood cell; JVP = jugular venous pressure.

Congenital Heart Diseases can be Acyanotic & cyanotic.

Acyanotic congenital heart disease

Atrial septal defect- Pregnancy is well tolerated by most women with an unrepaired atrial septal defect (ASD).

Paradoxical embolism and pulmonary hypertension are rare and arrhythmias uncommon in women younger than 40 years. Mitral regurgitation caused by mitral leaflet prolapse develops in up to 15% of cases of uncorrected ASD.

Acute blood loss at the time of delivery is poorly tolerated and can cause a large increase in left-to-right shunting; precipitous falls in left ventricular output, blood pressure and coronary blood flow; and even cardiac arrest.

Ventricular septal defect and patent ductus arteriosus

Ventricular septal defect (VSD) and patent ductus arteriosus (PDA) are well tolerated in pregnancy unless they are large or complicated by pulmonary vascular disease.

Pre-pregnancy evaluation of the presence of a defect, cardiac dimensions and estimation of pulmonary pressures are recommended.

Pulmonary stenosis

Pulmonary stenosis (PS) is well tolerated, although severe cases may precipitate right-sided heart failure, tricuspid regurgitation or atrial arrhythmia.

Women with a pre-pregnancy symptoms should be considered for balloon valvuloplasty or surgery before conception.

Aortic stenosis and bicuspid aortic valves

In women of childbearing age, the main cause of aortic stenosis (AS) is congenital bicuspid aortic valves (BAoVs).

Patients can be asymptomatic, even with severe AS. Significant obstruction results if the aortic valve area is smaller than 1 cm2 Women with BAoV are at increased risk of aortopathy (and are therefore at higher risk of dissection) and arrhythmias.

The aortic root and ascending aortic diameter should therefore be assessed before and during pregnancy. In such women, surgery before pregnancy should be considered if the aortic root is >50 mm in diameter. In pregnant women with severe AS, heart failure occurs in about 10% and arrhythmias in 3–25%.13 All women with symptomatic AS

(chest pain, syncope or pre-syncope) or asymptomatic AS but impaired left ventricular function on a pathological exercise test(without an appropriate increase in blood pressure or the development of ST- or T-wave changes) should be counselled against pregnancy, and valvuloplasty or surgery should be performed before pregnancy.

Medical treatment (with â blockers and diuretics) and restricted activities are indicated for patients who ndevelop signs or symptoms of heart failure during pregnancy. If medical treatment fails,

Either balloon aortic valvotomy or, rarely, valve replacement after early delivery by caesarean section are options.

Coarctation of the aorta

Most cases of Coarctation of the Aorta (CoA) may be first diagnosed during investigation for hypertension in pregnancy.

Women with unrepaired native CoA and those with repaired CoA but residual hypertension or aortic aneurysms have an increased risk of aortic rupture and rupture of an associated cerebral aneurysm during pregnancy and delivery. Any narrowing or pre- or post-stenotic dilatation or aneurysm formation should be assessed with magnetic resonance imaging prior to pregnancy.

Optimal treatment of hypertension (ideally with â blockers) is necessary, although aggressive treatment should be avoided.

Strenuous exercise should be avoided, as adequate blood pressure control may not be maintained during exercise, increasing the risk of cerebral haemorrhage or aortic dissection.

Normal delivery is usually possible, although severe CoA would warrant a shortened second stage of delivery.

Marfan's syndrome

About 80% of patients with Marfan's syndrome have some cardiac involvement, commonly mitral valve prolapse and regurgitation.Patients with Marfan's syndrome and a normal aortic root diameter have a 1% risk of aortic dissection or other serious cardiac complications during pregnancy.

As such, pregnancy, even in the absence of pre-existing disease, increases the Susceptibility to aortic dissection due to haemodynamic and hormonal changes. Dissection occurs most often in the last trimester of pregnancy (50%) or the early postpartum period (33%).

Women with progressive aortic root dilatation and aortic root dimension >4 cm and those with a family history of dissection or sudden death, even in the absence of a dilated aortic root, are at increased risk of aortic rupture or dissection.

Women with aortic roots >4.6 cm should be advised to delay pregnancy Until after repair or root replacement.

Outcome of pregnancy is usuallygood in women with minimal cardiac involvement and an aortic root <4 cm in pregnancy.

Management includes monthly echocardiography to assess the aortic root in those with cardiac involvement and â blockers for hypertension or aortic root dilatation. Vaginal delivery for those with stable aortic root is possible, but elective caesarean section with regional anaesthesia is recommended if the aortic root is enlarged or dilating.

Tetrology of Fallot

The association of severe right ventricular outflow tract obstruction with a large subaortic VSD and overriding aorta causes right ventricular hypertrophy and right-to-left shunting with cyanosis. Pregnancy is usually well tolerated in

Uncorrected cases, but subcutaneous low molecular weight heparin (LMWH) should be given to prevent venous thrombosis and paradoxical embolism.

Women who have had previous surgical correction and do well in pregnancy, although pulmonary regurgitation from previous correction of right ventricular outflow tract obstruction may lead to right ventricular failure.

Pulmonary hypertension

Pulmonary vascular disease, secondary to Eisenmenger's syndrome or lung or connective tissue disease or due to idiopathic arterial pulmonary hypertension, is extremely dangerous in pregnancy, with maternal mortality of 25–40%.

In cases of unplanned pregnancy, elective termination carries a 7% risk of death.

Women with pulmonary hypertension who have left-toright shunts are at lesser risk and may do well during pregnancy.

Management includes drugs such as Sildenafil and Bosentan, elective admission for bed rest, oxygen, thromboprophylaxis with LMWH and serial monitoring of fetal growth.

Most fatalities occur during delivery or during the first week after birth.

Nebulised or intravenous prostacyclin can be used to prevent pulmonary vasoconstriction, although resuscitation is rarely successful when sudden deterioration occurs.

All cases should be discussed with a centre specialising in pulmonary hypertension.

Postoperative congenital heart disease

Survivors of neonatal palliative surgery for complex congenital heart disease need individual assessment.

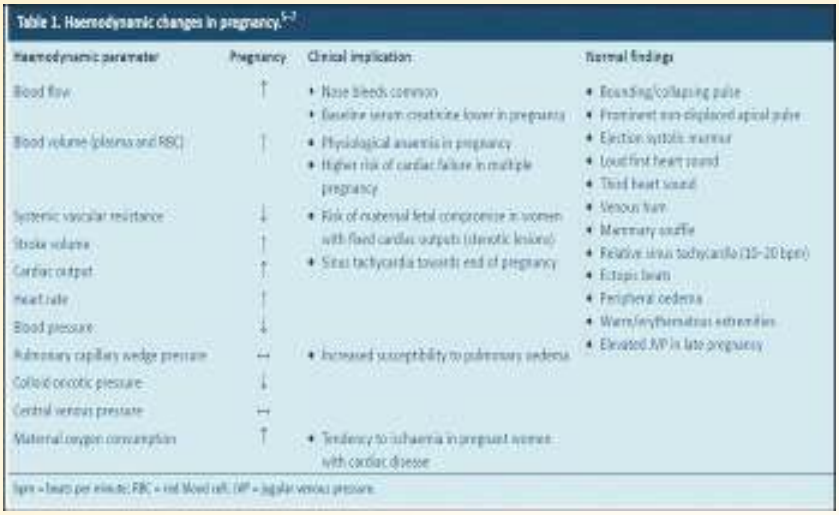
Following the Fontan operation for tricuspid atresia or transposition with pulmonary stenosis, the left ventricle provides the pump for both the systemic and pulmonary circulations.

Increases in venous Pressure can lead to hepatic congestion and gross oedema, but pregnancy

can be successful.

Anticoagulation with LMWH and optimal hydration peripartum are recommended to enable adequate left ventricular preload.

Table - 8



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bpm = beats per minute; RBC = red blood cell; JVP = jugular venous pressure.

Table 9. The Pregnancy Heart Team ACOG Guidelines[7]

	Modified WHO Pregnancy Risk Classification I	Modified WHO Pregnancy Risk Classification II	Modified WHO Pregnancy Risk Classifications III and IV
Pregnancy Heart Team Members	Obstetrician, family medicine practitioner, internist	Obstetrician, family medicine practitioner, internist Maternal-fetal medicine Subspecialist Cardiologist Consultation	Obstetrician, family medicine practitioner, maternal-fetal medicine subspecialist, internist, obstetric anesthesiologist, cardiology subspecialistsin adult congenital/ aortopathy*, heartrhythm*, heart failure*, pulmonary hypertension*, and cardiac imaging*Interventional cardiologist* Cardiac surgeon* Neonatologist*Geneticist* Mental health specialist*Pharmacist*

Interpretation and indications of Tests for the pregnant patient with possible heart disease -

Cardiac status testing is crucial for pregnant or postpartum women with symptoms like shortness of breath, chest pain, or palpitations and those with known cardiovascular disease. Risk assessment is based on family history and underlying medical conditions.

Conclusion:

Pre Pregnancy prediction of risks for both mother and child should be done, Available investigations have their limitations so a proper clinical judgement and multidisciplinary team apporach is mandatory.

The modified WHO classification is the first step in guiding management and followup during pregnancy, labour and post delivery.

Counseling should be done explaining the maternal and foetal risks,evaluation of medication and mode of delivery.

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"God could not be everywhere,
and therefore he made mothers."

–Rudyard Kipling

"Motherhood has a very humanizing effect.
Everything gets reduced to essentials."

–Meryl Streep

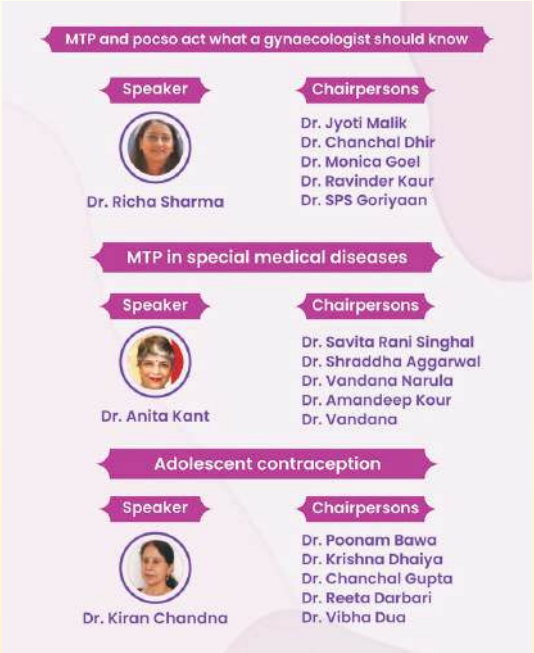
Kanoon ki Pathshala

- North India Gynecologist Forum is committed for Sensitizing all Ob & Gyn for potential medicolegal issues & cases.
- Dr Arun Arora (Jammu) & Dr Taru Chhaya (Jaipur) delivered deliberations on Case Record Keeping & Documentation prior to cesarean section.
- Sessions were followed by reverse panel on real legal cases and expert opinions.
- Dr Sangeeta Gupta & Dr Sadhana Gupta as convenor & Dr Sharda Jain as Patron is planning these sessions with meticulous efforts.

NIGF Chandigarh- Webinar on Female Sexual Dysfunction, 27th Jan, 2024

- We heartily Congratulate Chandigarh Chapter of NIGF for inaugural academia program.
- Subject was untouched area of female sexual dysfunction & we had International Faculty for it

NIGF Haryana Chapter 17th Feb 2024



- Great effort by Dr Ruby Bhatia, Dr Meenakshi Chauhan, Dr Richa Sharma, Dr Kiran Chandana & Team
- NIGF heartily congratulate Haryana Chapter with its collaboration with FOGSI MTP committee for an insightful program on intricacies of MTP laws and difficult case situations in MTP

NIGF Rajasthan Chapter 29th March 2024

- Hearty Congratulations for excellent academic program by Rajasthan Chapter of NIGF. Esp Congratulations Dr Veena Acharya, Dr Monika Gupta & Dr Taru Chhaya.
- PCPNDT act and Diagnosis of Birth Defect was well chosen subject.

Launch of Video film on updates in POCSO Act- 14 Feb 2024

- It was pride & pleasure to release video film on POCSO act on auspicious day of Vasant Panchami Under aegis of NIGF & DGF. Hearty Congratulations Dr Sharda Jain & Dr Sangeeta Gupta along with Dr Kiran Agrawal, Dr Gaurav Agrawal for their hard work.
- Members can access to whole video film on NIGF Facebook page https://www.facebook.com/groups/1356723371522327

NIGF Expert Speaks Series 1- 27th Mar, 2024

- NIGF First Episode of Expert speaks was successfully accomplished with great deliberation by Dr Smita Ramchandran on enigmatic subject of Precocious Puberty Don't miss opportunity to treat well in such cases as time is everything.
- Thanks Our Dignitaries, expert & panel for highly interactive session
- We are all set to next expert speaks series on Art of Non Descent Vaginal Hysterectomy by Dr Geeta Jain

NIGF Jammu Chapter & MTP Committee FOGSI inaugural Webinar 16th March 2024

- Jammu Chapter of NIGF started its Academic Series in collaboration with FOGSI MTP committee with discussions on newer approaches in second trimester MTP and legal and safety issues in MTP.
- Hearty congratulations to Dr Arun Arora & Dr Meeta Gupta along with Dr Richa Sharma & Dr Kiran Chandana.

- NIGF joining with FOGSI Female breast Committee for Breast Disease & crossing barriers to breast feedings.
- A well planned program on issue of great public health importance.

NIGF Punjab Chapter Webinar - 9th April 2024

- Punjab Chapter of NIGF organized Webinar on POCSO / MTP in special situations & contraception in collaboration with MTP committee on 9th April 2024

Platinum Oration
26th Jan

- Thanks & hearty congratulations for great republic day celebration Blessings by our mentor, Patron, Wonderful oration by Prof Veena Acharya Pearls of wisdom by all chairs Great judging & great participation & heart throbbing anchoring by Dr Mala & Dr Anita & release of NIGF Bulletin by Dr Jaya with inspiring poem. It was true Republic Day in spirit thought & action.

Precis Writing Competition 26th Jan 2024

- How we dream for Developed India – It was precis writing competition on our Republic Day
- Great thoughts wonderful presentation by our members
- We will carry many doable things forward under aegis of NIGF
- Hearty Congratulations to all winners & participants

NIGF Precis Writing Contest		
Rank	Name	Final scoring Sheet
1	Dr Taru Chhaya	185
2	Dr Anu Arora	177
3	Dr. Sarita Gupta	176
4	Dr Nishi Gupta	175.5
5	Dr Garima Goswami	175

1st Winner
Dr Taru Chhaya

Developed India of my Dreams

Trust the Magic of New Beginning

I love my India and want it to be the best in the **Panch-tatvas.**

1. SONE KI CHIDIYA. Cultivating varied healthy crops even in the barren lands, we would be in a position of exporting rather than importing.

2. SWACHH HI UCHCH. Even rural areas would have clean water supply and Sanitization. Well Equipped Hospitals would fulfil demands of the earliest. Anaemia and Cervical cancer free India is my dream. How I wish if all cancers had vaccines!!

3. SASHAKTI HI SAHI BHAKTI. Ideal Bharat would be when mentions of poverty and starvation are rare, women are independent and take up financial family responsibilities. Deserving candidates would excel indifferent to their caste.

4. NA BHRUSHT NA KASHT. When all abide by honesty from the grass root level. Zero tolerant matters would be considered so.

5. JO PADH GAYA SO TAR GAYA. Education would be compulsory till class 10 with higher education as per interest and ability, helping them reach their full potential and build a progressive India.

My dream if fulfilled, India will only be **UNSTOPPABLE**.

Dr Taru Chhaya
9829256161
Bansal Hospital
Shyam Nagar Jaipur

2nd Winner
Dr Anu Arora

Dashboard for Developed India of my dreams '

-I envision Developed India or 'Bharat Mata' as we fondly address, where all women will receive respect, untouched by gender bias or dowry pressures. Development cannot be perceived until each woman can walk safely on road without the fear of harassment.

-It will be pollution free green country where roads will be adorned with trees and devoid of litter.

-It will be an educational hub, reclaiming the glory of Nalanda University! Foreign Students will vie for our IITs and IIMs, and no student will travel ashore for higher education.

-Medical tourism will increase where India will have solution to all the ailments.

-Technologically ISRO will be ahead of NASA in all space missions and solve the enigma of various planets.

- In short My Developed India will be progressive and harmonious echoing the spirit of Ram Rajya once again! It will relive the title 'Sone ki Chidiya', providing basic amenities to all.

Dr Anu Arora

Jalandhar

3rd Winner
Dr. Sarita Gupta

DEVELOPED INDIA OF MY DREAMS- FOREMOST UNITY IN DIVERSITY

- Country being very vast with different languages, culture, religion, physical appearances due to this daily fighting, blame game, hatred amongst us should be stopped or minimized.
- Encouragement for more and more improvement in Technology & I.T. Sector is a must. It will improve economic status of an Indian.
- Education should reach the lowest strata of citizen.
- Health needs to be given priority. Preventive health care through Vaccines, Nutrition, and awareness about health hygiene & Aayushman scheme.
- Development of good Infrastructure including Roads, railways, communication
- Improve road connectivity for timely health care.
- Promote swach Bharat for better environment to enable good health and mental status.
- Accord priority for the defense of our Country so that nobody dares to attack India and our sovereignty is maintained.

This will ensure that name of my country is taken with respect in international forums. I wish to be a proud Citizen of India

Dr. Sarita Gupta

4th Winner
Dr Nishi Gupta

India of my dream would be an ideal country that sees all citizens as equal and does not discriminate them on any of criteria, a nation where every individual is able to reach their full potential and without any caste based reservation

1-Education should be necessary for each member.

2-women should be empowered and given equal rights

3-Our India should be clean and green, at every municipality level dirty areas should be developed such that more employment and better environment is created

4- lots of waste govt lands can be used for making schools, malls and hospitals under public and private partnership that can create miracles

5- Tourism should be promoted more as we have valley of flowers in Uttarakhand, Pahalgam and many unexplored marvellous architectural temples which are not even known to Indians

6- Pollution free clean and green

7- Digital India

Lastly most importantly is the reform in medical services which I can not describe due to words restriction.

I want India to be scientifically advanced, technologically better and agriculturally proactive.

For all these we should prevent brain drains of our intellectuals to other countries by giving them good opportunities and better working atmosphere

Dr Nishi Gupta
Vice President SOGA
Ambala, Haryana
9466658850

5th Winner
Dr Garima Goswami

Dashboard for developed India of my dreams

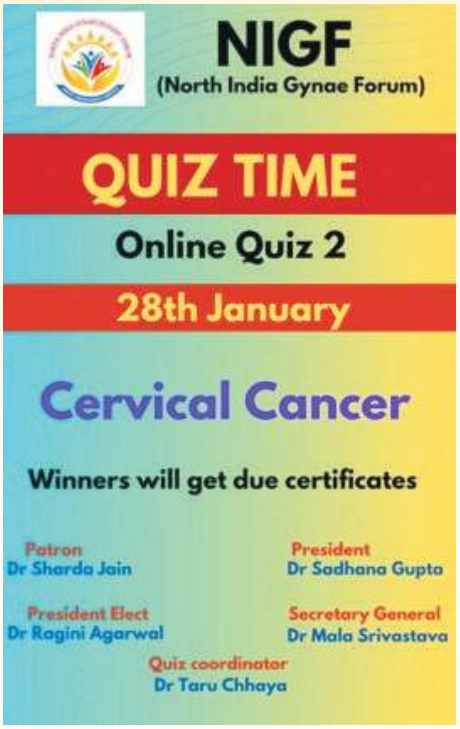
- It's foundation will be unity and respect for all. Peace will prevail and harmony will be celebrated with no discrimination.
- It will have all the latest technologies in use. Research based problem oriented approach to solve nationwide crisis.
- Quality Healthcare at affordable prices accessible for all. Population control with awareness about family planning and effective screening and treatment for all ailments. Dawn of Health awareness among the community.
- India will emerge as a world leader.
- The literacy rate will be 100% with reduced poverty rate.
- Improved sex ratio with gender equality.
- Use of robotics in all fields. Advanced equipments making life easy.
- Greater appreciation of our culture and values.
- Economic boost with increasing GDP and powerful financial position in the world.

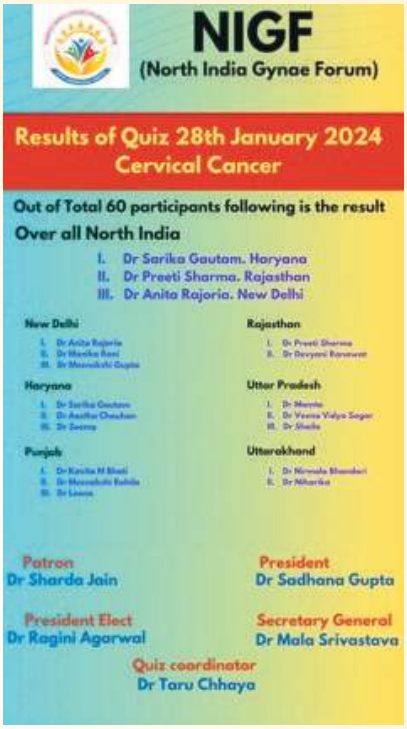
Click to add subt

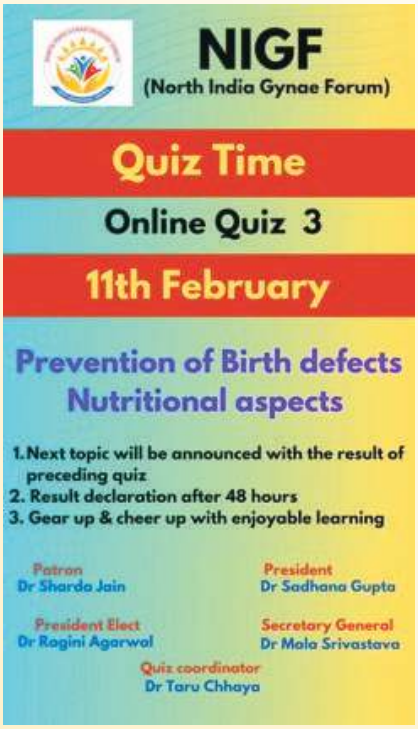
-Dr. Garima Goswami
M.B.B.S intern
M.M.I.M.S.R
M.M.U
Mullana Ambala

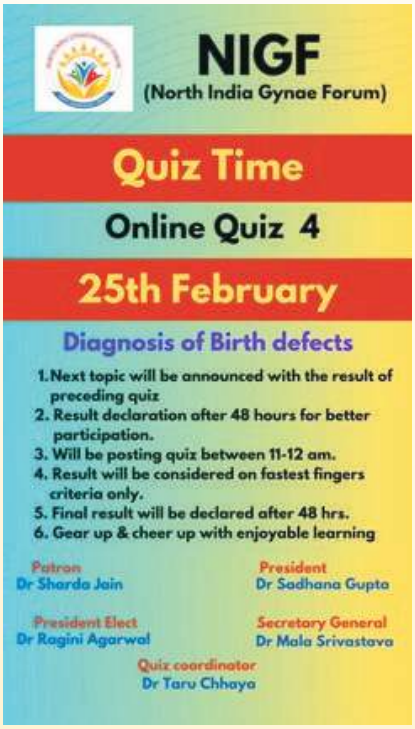
Quiz

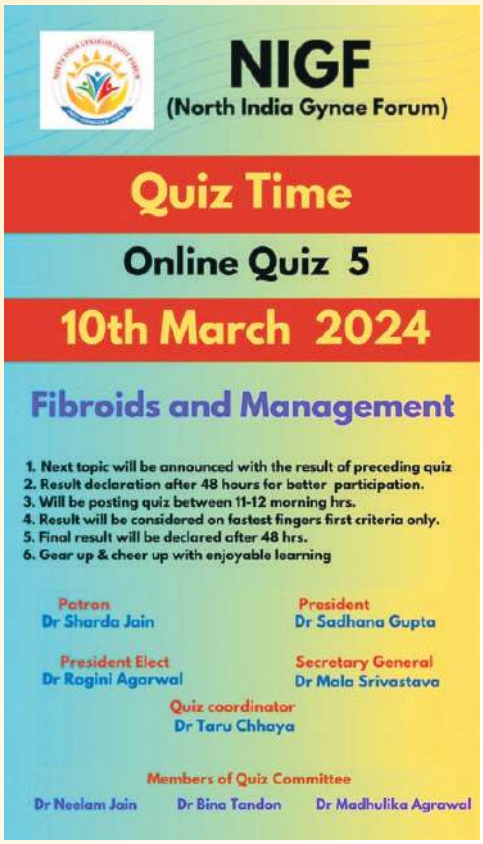
- Quiz Time Learning Time
- Every 2nd & 4th Sunday NIGF Quiz Team presents mind teasers on diverse subjects to our members under able coordination of Dr Taru Chhaya
- Fastest and highest scorers receive certification
- Keep on Enjoyable Learning



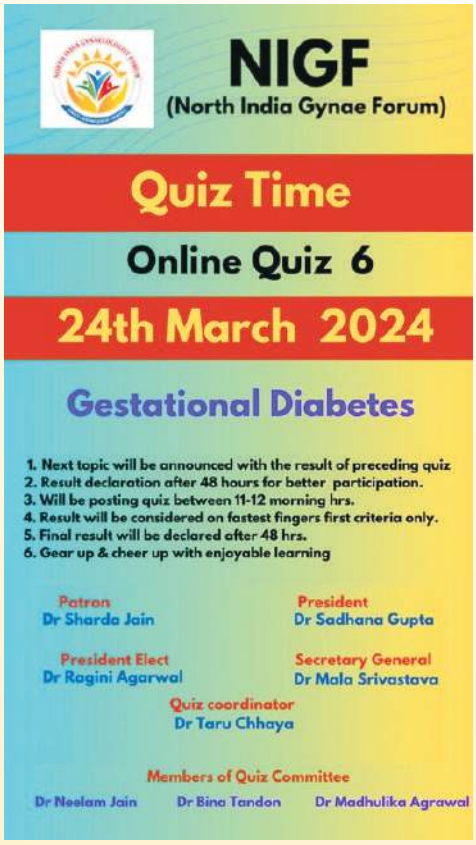







NIGF
(North India Gynae Forum)
Quiz Time
Online Quiz 5
10th March 2024
Fibroids and Management
1. Next topic will be announced with the result of preceding quiz
2. Result declaration after 48 hours for better participation.
3. Will be posting quiz between 11-12 morning hrs.
4. Result will be considered on fastest fingers first criteria only.
5. Final result will be declared after 48 hrs.
6. Gear up & cheer up with enjoyable learning
Patron
Dr Sharda Jain
President
Dr Sadhana Gupta
President Elect
Dr Ragini Agarwal
Secretary General
Dr Mala Srivastava
Quiz coordinator
Dr Taru Chhaya
Members of Quiz Committee
Dr Neelam Jain
Dr Bina Tandon
Dr Madhulika Agrawal


NIGF
(North India Gynae Forum)
Results of Quiz 5 , 10th Mar 2024
Fibroids anf management
Out of Total 200 participants following is the result
Over all North India
I. Dr Vandana Gupta Delhi. 10/10
II. Dr Gaurav Gupta Delhi 10/10
III. Dr Meenakshi Delhi 10/10
New Delhi
I. Dr Vandana Gupta Delhi. 10/10
II. Dr Gaurav Gupta Delhi 10/10
III. Dr Meenakshi Delhi 10/10
Haryana
I. Dr Smriti Anand. 9/10
II. Dr Latika Duhan 8/10
III. Dr Nidhi Oberoi 7/10
Punjab
I. Dr Leena 10/10
II. Dr Sukhman 10/10
III. Dr Ratinder Kaur 9/10
Rajasthan
I. Dr Charu Johri 10/10
II. Dr Preeti Sharma 7/10
III. Dr Monika Gupta 7/10
Uttar Pradesh
I. Dr Seema Saran 10/10
II. Dr Poole Srisala 10/10
III. Dr Mamta Rajan 9/10
Uttarakhand
I. Dr Priyanka Chauhan 6/10
II. Dr Anila Gupta 6/10
III. Dr Usha Rawat 6/10
All over 20 participants secured 10/10.... Congs....
Patron
Dr Sharda Jain
President
Dr Sadhana Gupta
President Elect
Dr Ragini Agarwal
Secretary General
Dr Mala Srivastava
Quiz coordinator
Dr Taru Chhaya
Members of Quiz Committee
Dr Neelam Jain
Dr Bina Tandon
Dr Madhulika Agrawal


NIGF
(North India Gynae Forum)
Quiz Time
Online Quiz 6
24th March 2024
Gestational Diabetes
1. Next topic will be announced with the result of preceding quiz
2. Result declaration after 48 hours for better participation.
3. Will be posting quiz between 11-12 morning hrs.
4. Result will be considered on fastest fingers first criteria only.
5. Final result will be declared after 48 hrs.
6. Gear up & cheer up with enjoyable learning
Patron
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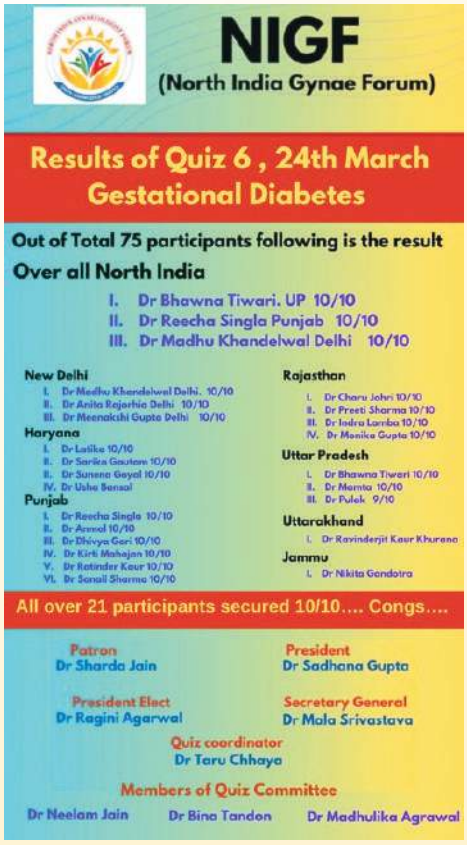
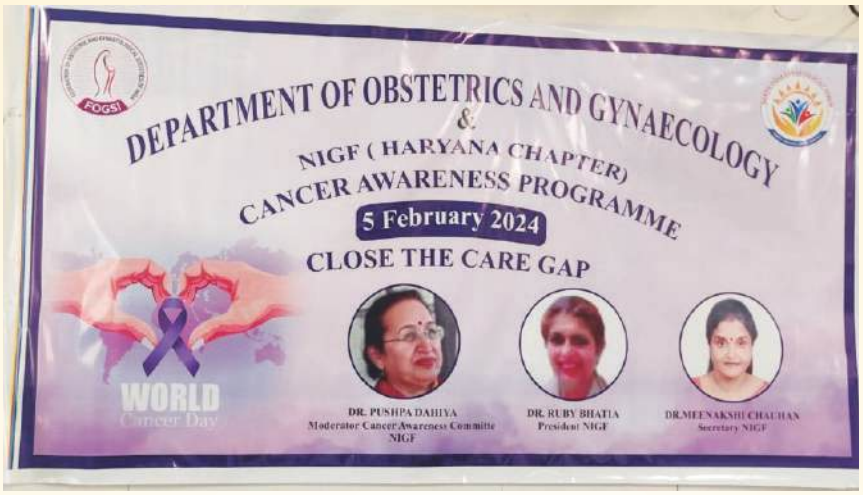
Dr Neelam Jain
Dr Bina Tandon
Dr Madhulika Agrawal


NIGF
(North India Gynae Forum)
Results of Quiz 6 , 24th March
Gestational Diabetes
Out of Total 75 participants following is the result
Over all North India
I. Dr Bhawna Tiwari. UP 10/10
II. Dr Reecha Singla Punjab 10/10
III. Dr Madhu Khandelwal Delhi 10/10
New Delhi
I. Dr Madhu Khandelwal Delhi. 10/10
II. Dr Anita Rajasthia Delhi 10/10
III. Dr Meenakshi Gupta Delhi 10/10
Haryana
I. Dr Latika 10/10
II. Dr Sarika Gautam 10/10
III. Dr Sunena Goyal 10/10
IV. Dr Usha Bansal
Punjab
I. Dr Reecha Singla 10/10
II. Dr Arvind 10/10
III. Dr Divya Gori 10/10
IV. Dr Kirti Mahajan 10/10
V. Dr Ratinder Kaur 10/10
VI. Dr Sonali Sharma 10/10
Rajasthan
I. Dr Charu Johri 10/10
II. Dr Preeti Sharma 10/10
III. Dr Indra Lamba 10/10
IV. Dr Monika Gupta 10/10
Uttar Pradesh
I. Dr Bhawna Tiwari 10/10
II. Dr Mamta 10/10
III. Dr Pulak 9/10
Uttarakhand
I. Dr Ravinderjit Kaur Khurana
Jammu
I. Dr Nikita Gandotra
All over 21 participants secured 10/10.... Congs....
Patron
Dr Sharda Jain
President
Dr Sadhana Gupta
President Elect
Dr Ragini Agarwal
Secretary General
Dr Mala Srivastava
Quiz coordinator
Dr Taru Chhaya
Members of Quiz Committee
Dr Neelam Jain
Dr Bina Tandon
Dr Madhulika Agrawal

- Hearty congratulations to Haryana Chapter of NIGF for conducting social program on prevention of cancer cervix, along with Nukkad Natak & poster competition Hearty congratulations



- Cervical Cancer Awareness program was organised by the Department of OBGYN, CMC Ludhiana under the aegis of NIGF on 1/2/24. This included talks on Cervical cancer prevention and screening. A role play on cervical cancer awareness was enacted by the nursing staff. A free PAP smear camp is being conducted by the department from 29th January to 4 th February 2024

- A Public awareness program on Breast and Cervical Cancer was held at PEACE Foundation, Ludhiana on 9th February 2024 by CMC Ludhiana under the aegis of NIGF. The talks included awareness, prevention and screening methods for breast cancer and cervical cancer. There were 61 participants. The speakers on the occasion were Dr Kavita M. Bhatti and Dr. Sharanpreet Kaur .

- NIGF & DGF organize a full day Hybrid CME (18 Feb 2024) on Hypertensive Disorders in Pregnancy with scientific deliberations by experts Pan India & workstations on Diverse practical issues
- Meeting was attended by 150 plus participants
- Hearty congratulations for Dynamic leadership of Dr Sharda Jain and able team of Dr Meenakshi Ahuja & Dr Shashi Kabra Maheshwari.
- Highlight is that all lectures were compiled in form of E book which was delivered to members.








- Awareness program on safe abortion and contraception with inner wheel club under dynamic leadership of Dr Monika Gupta Secretory Rajasthan Chapter of NIGF

- Celebrating women's day at Rajeev Gandhi Chawk (CP) in Delhi on Sunday 10th April by Delhi Chapter of NIGF, Hearty Congratulations Dr Meenakshi Ahuja & Dr Shashi Kabra Maheswari.

BLOCK
YOUR DATES

for

2nd NIGF Annual Conference

Dates : **27 & 28 July 2024**

Venue : **Hotel Radisson Blu**, Gorakhpur

Theme-

Holistic Reproductive Health

Please write to
drguptasadhana@gmail.com
for your area of interest

We wish our members

Happy Safe Motherhood Day

11 April