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A Brief Summary of Complications during Pregnancy and Their Management

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Abstract: *Pregnant women have an increased morbidity and mortality for certain illnesses owing to the physiologic and immunological changes in pregnancy. The most common complications include gestational diabetes mellitus, pre eclampsia, hyperemesis gravidum, infections, anemia. The management of women with these complications requires special attention and understanding the effects on pregnancy. To prevent these complications , pregnant women should be frequently monitored. Gestational diabetes mellitus carries an increased risk of metabolic disease in the mother and child. The gold standard treatment for gestational diabetes is insulin and oral hypoglycaemic agents(metformin, glyburide). Pre eclampsia is a multisystem disorder of unknown etiology usually associated with elevated blood pressure and proteinuria. Adequate and proper prenatal care is the most important part of management of pre eclampsia. Bacterial vaginosis is the most common lower genital tract syndrome in women of reproductive age. Urinary tract infections are one of the most common complications of pregnancy. When the lower UTI's of asymptomatic bacteriuria and cystitis are not eradicated, the subsequent risk of the development of pyelonephritis is increased. Women often become anemic during pregnancy because the demand for the iron other vitamins is increased due to physiologic burden of pregnancy. The inability to meet the required level for these substances is either as a result of dietary deficiencies or infections give rise to anemia.*

Key Words: *Pregnancy, Urinary tract infection, Gestational diabetes mellitus, Pre-eclampsia*

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I. Introduction

A complication of pregnancy occurs during the period from conception. Pregnancy affects most organ systems (neurological, respiratory, cardiovascular, hematological, and renal). Common minor health issues include constipation, gastro esophageal reflux disease (GERD), incontinence, edema and nausea/vomiting have been treated safely and effectively using selected drug therapies. Various acute and chronic illnesses may cause risk to women and the fetus. The identified illness should be treated with appropriately selected and monitored drug therapies. Most of these major complications are seen in second and third trimester of pregnancy. All these physiological changes appear to be endogenous and regulatory mechanisms helpful for the mother to tolerate stresses of pregnancy, labor, and delivery[1].

Stages of pregnancy	Complications	Causes
1st trimester	(A) Hyperemesis gravidum (Characterized by severe nausea, vomiting, dehydration and weight loss)[2].	UTI, high thyroid levels, hormonal changes, multiple pregnancies, family history of HG.
	(B) Vaginal bleeding [3].	Spontaneous abortion, miscarriage, low birth weight, preterm delivery, hydatidiform mole, ectopic implantation [3].
2nd trimester	(A)Preterm labor	Weak cervix, ante partum hemorrhage, hypertensive disorders of pregnancy.
	(B) Preterm premature rupture of membrane (PPROM)	Uterine infection, Over stretching of uterus and amniotic sac, trauma.
	(C) Cervical incompetence	Malformed cervix or uterus from a birth defect, previous surgery on the cervix, DES (Diethylstilbestrol) exposure.
	(D) Preeclampsia[2]	Insufficient blood flow to the uterus, prior history of hypertension, history of obesity.
		Obstetric complications, such as amniotic fluid embolism, chorioamnionitis,

	(E) Respiratory problems	trophoblastic embolism, and placental abruption, can produce acute lung injury
	(F) Miscarriage	Due to various factors (injury, drugs, physiological changes) other than chromosomal.
	(G) Gestational diabetes	During pregnancy, the placenta produces a hormone that leads to raised blood glucose.
	(H) Bleeding gums	Due to increased blood flow
	(I) Oligohydramnosis	uteroplacental insufficiency, drugs, fetal abnormalities, or premature rupture of membranes
3rd trimester	(A) GERD	Medications (aspirin), alcohol consumption, spicy foods, large meals, and irregular meals.
	(B) Preeclampsia	Carrying more than one fetus, age more than 35 or older [4].
	(C) Placenta previa	
	(D) Gestational diabetes	Multiple pregnancies.
	(E) Intrauterine growth restriction	Infection, antiphospholid Syndrome, genetic problems or a congenital anomaly in the baby, pre-existing medical problems in the mother

Table 1- Types of complications based on the stages of pregnancy.

The most common complications of pregnancy includes anemia and infections, these complications can be seen in all stages of pregnancy.

This article provides a brief summary on major complications includes gestational diabetes mellitus (DM), preeclampsia, hyperemesis gravidum, oligohydramnosis, infections and anemia.

II. Gestational Diabetes Mellitus (Gem)

GDM is defined as carbohydrate intolerance of varying degrees of severity with onset or first recognition during pregnancy. The prevalence of GDM is increasing worldwide. Yearly 21millions of GDS cases (7%of population) are reported [5]. According to WHO, the standard screening test for GDM has proposed using a hour 75gm oral glucose tolerance test (OGTT) with a threshold plasma glucose concentration of greater than 140mg/dl at 120min[6]. According to studies the major risk factors associated with GDM were age of the patient, poly cystic ovarian disease (PCOD), previous and family history of GDM, previous history of macrosomic baby [7].

Mechanism involved in GDM is, generally insulin sensitivity decreases in pregnant women which leads to maternal hyperglycemia. In non pregnant state insulin concentrations are raised about three times. Free cortisol and pregnancy associated hormones (progesterone, estrogen, luteinizing hormone) causes post insulin receptor events (by cellular effects) which leads to increased insulin resistance. The major causes of insulin resistance are genetic deficiency of glycogen synthase activation, compounded by additional defects due to metabolic disorders, receptor down regulation, and glucose transporter abnormalities, all contributing to the impairment in muscle glucose uptake.

Maternal complications include still birth, increased frequency of congenital abnormalities. Whereas fetal complications include macrosomia (20-30%), hypoglycemia, jaundice, respiratory distress syndrome, polycythemia and hypocalcaemia [8].

Treatment for GDM

Most of the oral hypoglycemic agents are not recommended in pregnancy. Some studies supports the use of glyburide (pregnancy C category) and metformin (pregnancy B category)[9].'

A. Metformin

Glycemic derangement	Start dose	Timing of dose	Adjustment of dose
Elevated fasting	500mg	HS	Every 3days as tolerated, increase dose by 500mg until
Normal fasting/ elevated fasting	500mg	With breakfast or dinner	

Both fasting and post meals are elevated	500mg BD	HS and breakfast or dinner	therapeutic effect or maximum of 250mg is reached then add insulin[10].

Table-2 Dose adjustments of metformin.

B. Glyburide

Glycemic derangement	Start dose	Timing	Adjustment
Elevated fasting alone	1.25 mg/day (maternal body weight <200lb) Or 2.5mg (maternal body weight >200lb)	HS	Every 2-3 days increased by 1.25mg until glycemic goals achived or 20mg/day maximum is reached
Elevated post meal	1.25 mg/day (maternal body weight <200lb) Or 2.5mg (maternal body weight >200lb)	30-60min pre meal	Every 3-7 day increased by 1.25mg until glycaemic goals achieved or until maximum dose of 20mg/day[10].

Table-3 Dose adjustments of glyburide.

C. Insulin

It is the gold standard treatment for GDM.

Weeks gestation	Total daily insulin
Week 1-18	0.7U/kg
Week 18-26	0.8U/kg
Week 26-36	0.9U/kg
Week 36-40	1.0U/kg[11]

Table-4 Recommendations of insulin based on gestation weeks

Intial insulin therapies	with mild hyperglycemia
Glycemic derangement	**Suggested insulin type and dose**
Persistent FBG >95mg/dl <120mg/dl.	Start 8-20U NPH at bedtime (0.165/0.2/kg) actual body weight.
One hour post breakfast plasma value >135mg/dl <180mg/dl.	Start 2-4 U Lispro or Aspart pre breakfast.
One hour post lunch plasma value >135mg/dl <180mg/dl.	Add 8-10 U NPH to pre breakfast injection (and eat lunch 4-5hrs after breakfast) or give 2-4 U Lispro or Aspart pre lunch.
One hour post dinner plasma value >135mg/dl <180mgdl.	Given 2-4 U Lispro or Aspart pre lunch[12].

Table-5 Initiation of insulin therapy based on glycemic derangements.

III. Pre-Eclampsia

Preeclampsia is defined as a condition in pregnancy characterized by abrupt hypertension (HTN), albuminuria and edema of hands, feet and face. It is the most common complication of pregnancy, usually occurs in 2nd trimester. Based on early onset (before 32-34 wks of pregnancy) of preeclampsia and fetal morbidity, preeclampsia is classified as mild / severe (severe HTN, severe proteinuria or substantial maternal organ dysfunction).

In India, the incidence of preeclampsia is reported to 8-10% among pregnant women. In India, the prevalence of hypertensive disorders of pregnancy was 7.8% and preeclampsia in 5.4%. Around world, the incidence of preeclampsia is 3.5% [13].

The major causes of preeclampsia are disturbances in placentation followed by inflammation and progressive endothelial damage. Other causes include premigravida, obesity, having a history of preeclampsia, chronic HTN/ kidney disease during pregnancy and multiple pregnancies.

Mechanism involved in preeclampsia is, normally, in preeclampsia reduced uteroplacental perfusion which leads to abnormal cytotrophoblast invasion of spinal arterioles. The dysfunction of maternal vascular endothelium by placental ischemia/ hypoxia results in enhanced formation of thromboxane, endothelin, superoxide, increased vascular sensitivity to angiotensin II and decreased formation of vasodilators such as nitric oxide (NO) and prostacyclin. These endothelial abnormalities cause hypertension by renal function and increased in total peripheral resistance. The potential mediators of endothelial dysfunction are cytokines and other factors like lipid peroxides and reactive oxygen intermediates which causes vasoconstriction results in elevated pressure during pre-eclampsia[14].

IV. Treatment for Pre-Eclampsia

Mild –Moderate pre-eclampsia: It is defined as blood pressure with systolic level of 140-159mmHg and diastolic of 90-109mmHg. Safe agents include methyldopa, labetalol, nefidipine. Most of the guidelines prefer labetalol (expect when contraindicated) to prevent the risk of hemorrhagic stroke[15].

a. Methyldopa
Initial dose – 250mg PO q 8-12hr for 2days, increased q 2days PRN.
Maintenance dose – Oral: 250-1000mg/day divided q 6-12hr PO, usually not more than 3g/day.
IV : 250-1000mg infusion over 30-60minutes q 6-8hr PRN not more than 4gm/day.

b. Nefidipine
30-60mg PO once daily may be increased every 7-14days PRN, not to exceed 120mg/day.'

c. Labetalol
Initial dose – 100mg PO q 12hr, increased by 100mg q 12hr every 2-3 days.
Maintenance dose – 200-400mg PO q 12hr.
Maximum dose – 2400mg/day.

Severe pre-eclampsia: It is defined as systolic pressure ≥160mmHg or diastolic pressure ≥110mmHg. Safe drugs include labetalol (IV), nefidipine, hydralazine (IV)'

a. Hydralazine
Oral: 25-100mg BD
IV: 5-10mg. A continuous infusion of 0.5-10mg/hr is recommended in critical conditions[16].

b. Labetalol
Initially 20mg by slow IV injection over a 2min period.
Magnesium sulphate is the first line agent for recurrent seizures (eclampsia). The regimen recommended for eclampsia is 4-5gm of magnesium sulphate IV over 5min, followed by an infusion of 1gm/hr for 24hrs. If recurrent seizures occur, an additional 2gm IV magnesium sulphate should be administered[17].

V. Hyper emesis Gravid arum (Hg)

HG is a condition with multifactorial etiology causing severe nausea and vomiting in the early pregnancy ultimately leading to metabolic disturbance such as carbohydrate depletion, dehydration, electrolyte imbalance and acid-base imbalance. Upto 80% of all pregnant women experience some form of nausea and vomiting during their pregnancy which doesn't have severe complications and found to have a lower risk for foetal loss and nonsyndromic oral clefts[18]. Women with HG experiences mechanical forces which results in the consequences of pneumomediastinum and esophageal rupture[19].

Etiopathogenesis

Risk factors include younger, primiparious, alcohol, smoking, body mass index, socio-economic status[20], major risk factor being hormonal imbalance. Paternal genes are not thought to play a role in the occurrence of HG. HG is associated with hormones like progesterone, estrogen, HCG produced by placenta and corpus luteum.

HCG – In conditions like twin and molar pregnancies, pregnancies of female fetuses and those with down syndrome have elevated HCG (specifically, HCG with asialo-carbohydrate chains) leading to the occurrence of HG[21]. The proposed mechanisms include stimulation of secretary processes in the upper GIT or by thyroid function.

Progesterone – The progesterone decreases smooth muscle contractibility and alters gastric emptying and lead to increased nausea and vomiting. Exogenous administration of progesterone for luteal phase support, alone does not increase the incidence of HG.

Estrogen – High estrogen (E_2,E_3) levels cause slower instestinal transit time and gastric emptying, and result in an increased accumulation of fluid caused by elevated steroid hormones. Change in p^H in the GIT could lead to the manifestation of subclinical H pylori infection, which could be related to GI symptoms[22].

Lower esophageal sphincter pressure (LESP) – Many women have symptoms of gastrointestinal reflux during their pregnancy which may result in progressive decrease in lower esophageal sphincter pressure.

Other common causes include gastric and intestinal motility, immunology, Helicobacter pylori infection, placental serum markers (schwangerschafts protein 1), growth hormone and prolactin, leptin, gestational transient thyrotoxicosis[23].

Treatment for Hyperemesis gravidarum

Non pharmacological therapy includes dietary changes (adapting protein rich foods[24] than carbohydrates and liquids than solids which improve gastric dysrhythmia associated with HG and emotional support (supportive psychotherapy, behavior therapy and hypnotherapy). Others include acupuncture, ginger

which stimulates GIT motility and stimulating the flow of saliva, bile and gastric secretion and it also has similar activity as $5HT_3$ antagonist.
Pharmacological treatment – In most of the patients, IV fluid therapy, vitamin supplementation, electrolyte imbalance correction are sufficient to relieve symptoms and prevent serious complications. When patients fail to respond, antiemetic therapy is also administered.

a. Vitamin supplementation – Pyridoxine (vitamin B_{16}) in combination with 10mg of doxylamine.
b. Promotility agents – 10mg of metachlopromide given every 8hrs to reduce the number of vomiting episodes.
c. Anti-emetics – Ondansetron: 10mg IV q 8hr PRN. Promethazin: 25-50mg IV q 8hr.
d. Antacids containing aluminium or calcium are first line agents for acid reflux and heart burn[25].
e. Nutritional support – Patients who are unresponsive to dietary modifications and pharmacological treatment and unable to maintain oral intake, nutritional support might be required. Intravenous fluids plays an important role in increasing the volume and restoring the electrolytes, IV thiamine should be administered before any dextrose containing fluids to avoid wernick's encephalopathy[26]. If IV therapy is not successful in reducing the symptoms, enteric tube feeding and total parenteral nutrition is considered.

VI. Infections in Pregnancy

1. SYPHILIS

It is a bacterial sexually transmitted infection caused by Treponema pallidum that results in substantial morbidity and mortality. It is transmitted through sexual contact with infectious lesions of mucous membranes or abraded skin, via blood transfusion or trasplacentally from a pregnant women to the fetus.
Treatment is based on syphilis screening or testing strategies :

A. Early syphilis (primary, secondary and early latent syphilis of not more than 2yrs duration) Benzathine penicillin G 2.4 MU once IM over no treatment (strong recommendation). Benzathine penicillin G 2.4 MU once IM over procain penicillin 1.2MU IM once daily for 10days (conditional recommendation). If these agents can not be used (because of unavailability or allergy) then, erythromycin 500mg orally four times daily for 14 days(or) ceftrioxone 1gm IM once daily for 10-14days (or) azithromicin 2gm once daily.

B.Late syphilis (infections of more than 2years duration without evidence of treponema infection) – Benzathine penicillin G 2.4 MU IM once weekly for 3 consecutive weeks over no treatment (strong recommendation). Benzathine penicillin G 2.4 MU IM once a day for 20 days, when it is not available, erythromycin 500mg orally 4times, daily for 30 days is usually preferred[27].

2. URINARY TRACT INFECTION

Urinary tract infections are one of the most frequent complications during pregnancy. Basically, UTI is classified as lower UTI (acute cystitis) and upper UTI (acute pyelonephritis). Asymptomatic bacteriuria is defined as a presence of positive urine culture in an asymptomatic person and occurs in 2-7% of all pregnancies, which is associated with increased risk of adverse fetal outcomes.

A. Asymptomatic bacteriuria – several studies have shown that the following regimens are effective in the treatment of asymptomatic bacteriuria in pregnancy.
Co-amoxiclav 625mg BD/ 500mg BD for 5days
Cefuroxime axetil /250mg BD/ 500MG BD for 5days
Cefaclor 500mg for 5-7days
Fosfomycin 3gm stat dose
Nitrofuraintoin 100mg BD PO for 7days

B. Lower UTI (cystitis) in pregnancy
Fosfomycin 3gm stat dose PO
Ceftibuten 400mg PO for 3days
Cephalexin 500mg BD/ TDS PO for 7days
Amoxicillin 500mg TDS PO for 7days
Additionally, the agents used to treat asymptomatic bacteriuria are recommended.

C. Upper UTI (pyelonephritis) in pregnancy
1. First line agents – Ceftriaxone 1-2gms daily until 48hrs afebrile then oral cephalexin 500mg QDS for 10days.
2. Second line agents –
(a) Clindamycin 900mg TDS/ vancomycin 1gm BD IV

(b) Gentamycin 1.5mg/kg TDS/ 5mg/kg OD IV until the patient is 48hrs afebrile then change to a PO alternative depending on susceptibility results and risk of teratogenicity.
3. Third line agents – Ciprofloxacin 750mg BD, PO for 7days.

3. BACTERIAL VAGINOSIS:
Bacterial vaginosis is the most common genital tract disorder among women of reproductive age and the most prevalent cause of vaginal discharge and malodour. Causative organisms include Gardnerella vaginalis, Mobiluncus species, Bacteriods and Prevotella species amd Mycoplasma species [28].

Oral – Clindamycin 300mg (or) metronidazole 500mg twice daily for 7 days

Topical – Clindamycin 5 gm (or) metronidazole at bed time for 5 days.

4. YEAST INFECTIONS:
Vulvovaginal candidiasis(VVC) is often referred as yeast infection and is a common gynecologic aliment. 80-90% of VVC is caused by Candida albicans and rarely by Candida glabrata and Candida tropicalis.
Antifungals – Fuconazole- 150mg Nystatin- 1,00,000 units intravaginally OD for 2 weeks
At higher doses(> 400mg/day) fluconazole has been accosiated with major mal formations.

Antiseptics – Boric acid-600mg intravaginally per night for 14 consecutive nights [29].

VII. Anemia

Definition: Hemoglobin less than 11g/dl; mild anemia when 9-11g/dl; moderate anemia when 7-8.9g/dl; severe when less than 7g/dl.

Anemia is the most common medical disorder in pregnancy which causes 2-3 fold increase in requirement of iron and 10-20 fold increase in folate requirement. In iron defiency anemia, there is a shortage of ferritin levels, reduced transport and functional iron limiting red cell production. Complications of anemia include preterm labour, physical weakness, increased risk of post partum hemorrhage, low birth weight babies and post natal depression.

Treatment:
Haematinics:
A. Ferrous sulphate-200mg PO 2-3 times per dayplus
B. Folic acid- 5mg PO once daily(also prevents neural tube defects specifically anencephaly and spina bifida)

For the treatment to be effective, the therapy should be based on hemoglobin levels.
1. Hb 80-100g/dl- if asymptomatic ang haemodynamically stable, offer 200mg elemental iron per day for 3 months. Full blood count and ferritin levels should be checked after 3 weeks to ensure that Hb and iron stores are repleted.
2. Hb <80g/dl- consider total dose intravenous iron. Repeat FBC and ferritin at 10 days to ensure response and at 3 months in community to ensure Hb and iron stores are repleted.
3. Hb <70g/dl- consider and discuss alternatives with the women. Consider blood transfusion and/or total dose intravenous iron. Give frusemide 40-80mg before blood transfusion. Continue with haematinics[30].

VIII. Discussion

Due to physiological changes in the pregnant women they are more prone to complications. These complications eventually leads to increased morbidity and mortality unless proper treatment is provided. Gestational diabetes and pre eclampsia are the most common complications seen in the pregnant women. To prevent the severity of these complications screening tests are usually performed for early detection. Appropriate treatment guidelines should be followed for treating the pregnant women with theses complications. The treatment choice should not effect the mother and fetus.

IX. Conclusion

This article provides brief information on the commonly occurring complications and their management in pregnant women. The usage of medications in pregnant women is limited as many drugs cause adverse outcomes (teratogenicity, increased risk of complications). Special measures and supportive care should be taken during treating the complications in pregnant women.

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