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SOCIETY OF  
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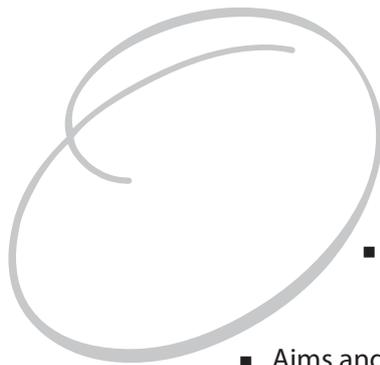
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Dear Members,

Welcome to second issue of our e-news letter. This issue focuses on renal diseases in pregnancy. First article elaborates on anatomical and physiological changes in urinary system in pregnancy and estimation of renal functions in pregnancy. Understanding physiological changes helps correctly interpret the renal parameters in pregnancy which are different from non pregnant state. Second article is a comprehensive review of chronic kidney disease in pregnancy which includes pre-pregnancy counselling of women with chronic kidney disease. This review also talks about common chronic kidney diseases encountered in pregnancy eg., Lupus nephritis, chronic pyelonephritis etc. Finally, a ready to use pro forma which can aid in planning management of pregnant women with chronic kidney diseases (follow up plan during ante natal period) has been included. Please feel free to copy the form and use it to help your pregnant women with kidney disease.

There has been a long interconceptional period between the first and second issues of e-news letter for various reasons! I request you to contribute articles for the next issue. We hope to bring out the next issue in April 2014 which will focus on tuberculosis and pregnancy. We invite articles related to 'tuberculosis and pregnancy'. You can submit interesting case reports, original articles, review articles and letter to the Editor with comments on previous articles. Your article will be reviewed by the editorial board before acceptance for publication. You can send in your articles to [obsmedindia@gmail.com](mailto:obsmedindia@gmail.com) with a copy to the Editor, [drharikishan@gmail.com](mailto:drharikishan@gmail.com). I also request you to encourage your colleagues and friends to join society of obstetric medicine, India.

Anticipating your contribution,

Dr. Hari Kishan Boorugu

# Physiological Changes in Urinary System in Pregnancy

– Dr. Devang Patel

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The prevalence of chronic kidney disease in women with childbearing age is 0.03 to 0.2% of all pregnancies (1). Although renal diseases in pregnancy is relatively uncommon; women with renal diseases are at increased risk for pregnancy related complications.(2,3) This review focuses on anatomical and physiological changes in pregnancy, common renal disorders encountered in pregnancy, effect of pregnancy on renal disease, effect of renal disease on pregnancy, pre pregnancy counseling and management.

It is important for the practicing clinician to be aware of the various physiologic and anatomic changes involving the urinary system that are induced by pregnancy.

## **Renal Anatomy in Pregnancy**

During pregnancy, the size of the kidneys increases by approximately 1 cm and the overall kidney volume increases by up to 30%. The urinary collecting system (renal calyces, pelvis, and ureters) dilates. Hormonal and mechanical forces are thought to account for ureteral dilation as early as 6 weeks gestation. (4). These morphological changes result in stasis in the urinary tract and a higher risk among pregnant women with asymptomatic bacteriuria for progression to pyelonephritis, particularly in those who have a history of prior urinary tract infections. (5)

## **Renal Physiology in Pregnancy**

Probably the most significant physiologic change is the increase in glomerular filtration rate (GFR) and renal plasma flow, which starts very early in pregnancy and exceeds non-pregnant levels by 50%. The GFR then falls by about 20% in last trimester and returns to pre-partum levels within 3 months of delivery. The increase in glomerular filtration, in turn, results in a significantly higher endogenous creatinine clearance (110–150 mL/min) and lower serum creatinine (0.5– 0.8 mg/dL) and serum urea nitrogen (9–12 mg/dL) levels.(3) The increase in GFR during normal pregnancy occurs without increase in intraglomerular pressure and normal pregnancy is not injurious to health.(1)

Glucose, water-soluble vitamins, protein and amino acids are excreted during normal pregnancy. This is attributed to the increase in GFR which causes the filtered load of nutrients to surpass the re-absorptive capacity of the

kidney with a consequent spill of these substances into the urine. (6) Total serum calcium levels fall in pregnancy but ionized calcium remains normal. Accelerated renal and placental production of calcitriol leads to increased gastrointestinal absorption of calcium and absorptive hypercalciuria with urine calcium as high as 300 mg/day. Serum parathyroid hormone (PTH) concentrations are lower than normal, partly in response to higher serum levels of calcitriol.

## **Acid – Base Regulation in Pregnancy**

Because of the increased circulating level of progesterone, which directly stimulates the medullary respiratory center, tidal volume and alveolar ventilation are increased during pregnancy, resulting in respiratory alkalosis. To compensate, the kidneys excrete more bicarbonate which results in 4 to 5 mEq/L decrease in serum bicarbonate to 20 to 22 mEq/L, changes that are apparent in the first trimester(7). A PCO<sub>2</sub> of 40 mm of Hg signifies considerable carbon dioxide retention in pregnancy.

## **Volume Regulation in Pregnancy**

Volume regulation in normal pregnancy is characterized by a gradual accumulation and retention of water and sodium. Most healthy women gain an average of 12.5 kilograms and most of this is fluid. The plasma volume doubles and this results in a fall in the plasma sodium concentration (dilution). Therefore, osmolality levels decrease. This would normally stimulate a diuresis by suppressing ADH. However, in pregnancy this does not appear to happen. It appears as though the osmoreceptors are 'reset' at a lower level to avoid a continuous diuresis.

Hyponatremia in pregnancy, may be caused partly by relaxin, a peptide hormone in the insulin family that is also associated with osmoregulatory changes and increases in GFR and vasodilatation in early pregnancy (8). Although serum sodium measurement decreases, a daily positive balance of 2 to 5 mEq and gradual accumulation of approximately 900meq of sodium is present during pregnancy (approximately 20g of sodium chloride). Serum potassium levels are normal despite increased serum aldosterone, perhaps due to the potassium-sparing effects of elevated progesterone levels in pregnancy.(4) The renin–angiotensin–

aldosterone system (RAS) is highly activated in normal human pregnancies, in response to vasodilation and a decrease in blood pressure. Plasma renin activity (PRA) increases 4-fold in the first trimester and continues to rise until approximately 20 weeks gestation.

### Normal Laboratory Values in Pregnancy (9)

Blood urea nitrogen (BUN), mg/dL	3 – 20
Creatinine, mg/dL	0.4 – 0.8
Creatinine clearance, mL/minute	50 – 166
Uric acid, mg/dL	2 – 4.9
Sodium, mEq/L	129 – 148
24-hour urine protein, mg	300
Arterial pH	7.39 – 7.45
PCo <sub>2</sub> , mm Hg	25 – 32
HCO <sub>3</sub> , mEq/L	18 – 22

### Assessment of Renal Function in Pregnancy

In non-pregnant population, the Cockcroft – Gault and MDRD (Modification of Diet in Renal Disease) formulae are most commonly used to assess kidney function. However, neither of these have been validated in pregnancy because of the changes in weight and body surface area.

Creatinine clearance measured with 24- hour urine collection remains the best approximate of the gold standard of inulin clearance and is the most well-validated method for measuring renal function in pregnancy.

The upper limit of normal for urinary protein excretion is 300 mg/dl in pregnant patients versus 150mg/dl in non- pregnant patients. Abnormal proteinuria has been evaluated with 24-hour urine collection, urine dipstick, and protein /creatinine ratio, but the gold standard remains the 24- hour urine protein measurement. A 24-hour protein level greater than 300 mg is abnormal in pregnancy and co relates with urine dipstick +1 protein measurement. Although commonly used in practice to detect significant proteinuria, urine dipstick testing is susceptible to error because of variation in urine

concentration. Total protein /creatinine ratio is of value in ruling out proteinuria if less than 250mg/ 24 hours but cannot be recommended as an alternative to 24- hour measurement as misclassification tend to occur when proteinuria is borderline (250-400mg/d).(10)

### Renal Disorders in Pregnancy

Acute	Acute Kidney Injury(Pre-eclampsia, HELLP , Thrombotic Microangiopathies)
	Acute Pyelonephritis
Chronic	Chronic Glomerulonephritis
	Focal glomerular sclerosis
	IgA nephropathy
	Diabetic Nephropathy
	Lupus Nephritis
	Polycystic Kidney Disease
	Chronic Pyelonephritis
Urolithiasis	

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## Chronic Renal Disease in Pregnancy

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Chronic renal disease can be classified based on the renal function into mild moderate and severe renal insufficiency.(1) The rationale is to identify those at high risk for poor maternal and fetal outcome.

### **Mild Renal Insufficiency**

Women with mild renal disease (i.e., serum creatinine 0.9–1.4 mg/dL) have a lower risk of adverse pregnancy outcomes than those with more advanced renal disease. The case series reported by Bar and associates (2) included 89% with mild renal disease. There were no stillbirths and the complication rate was very low (4.4–22%). It appears that the perinatal outcome for pregnant women with mild renal insufficiency is only slightly affected, and irreversible deterioration of the maternal renal function is uncommon.

### **Moderate-to-Severe Renal Insufficiency**

The rate of complications is clearly higher in pregnant women with moderate-to-severe renal insufficiency than in women with mild renal disease. Chronic hypertension, preeclampsia, anemia, fetal growth restriction, and prematurity are common complications in pregnant women with moderate-to-severe renal insufficiency. In the observational study by Cunningham and associates (3), there were 37 pregnancies complicated by chronic renal disease that was moderate (serum creatinine level 1.4–2.5 mg/dl) to severe (serum creatinine level greater than 2.5 mg/dl). In the 26 women with moderate renal disease, 62% had chronic hypertension, 58% had preeclampsia, and 73% had anemia. These 26 women had a live birth rate of 88% (23 of 26 women, excluding 2 spontaneous abortions and 1 stillborn), a fetal growth restriction rate of 35%, and preterm birth rate of 30%. In the 11 women with severe renal insufficiency, 82% had chronic hypertension, 64% had preeclampsia, and 100% had anemia. The fetal outcomes in these women with severe renal disease include a live birth rate of 64% (7 of 11 women, excluding the 2 elective abortions and 2 spontaneous abortions), fetal growth restriction rate of 43%, and preterm birth rate of 86%. Almost 50% of the pregnant women with a serum creatinine of 1.4 mg/dL or greater had an increase in serum creatinine during pregnancy to a mean of 2.5 mg/dL in the third trimester. The risk of accelerated progression to end-stage renal disease is highest when the serum creatinine level was above 2.0 mg/dl at the beginning of pregnancy.

Within 6 months after delivery, 23% of such women had progression to end-stage renal disease.

### **Management of Chronic Renal Disease**

#### **Pre-pregnancy or Early Prenatal Counseling**

Women with renal disorder is counseled that fertility is somewhat dependent upon the degree of renal insufficiency, as is pregnancy outcome.(4) Women with a serum creatinine level greater than 2.0 mg/dL should be counseled that they have a one-in-three chance of progressing to end-stage renal disease within 1 year postpartum. Women should be explained about the adverse maternal and fetal outcome that could occur depending on renal function. Pregnancy outcome is related to the degree of hypertension, the effectiveness of antihypertensive therapy, and the presence or absence of superimposed preeclampsia.

The need for blood pressure control and explanation that antihypertensive medications belonging to the angiotensin-converting enzyme inhibitor group (i.e., ACE inhibitors) and angiotensin receptor blockers are contraindicated during pregnancy because these drugs have the potential for causing teratogenic effects (hypocalvaria) and damage to fetal kidneys (renal failure, oliguria, and demise) is essential. Angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers should be discontinued before attempting pregnancy or as soon as possible in the first trimester after pregnancy is diagnosed.

#### **Management during Pregnancy**

Care for pregnant women with underlying renal disease should be under a multidisciplinary approach at a tertiary center and include a maternal-fetal medicine specialist and a nephrologist. It is advisable to follow such mothers every 2 weeks until 30–32 weeks of gestation and then weekly for the remainder of the pregnancy. In addition to the routine tests, baseline renal function should be assessed and then repeated at least every 4–6 weeks throughout pregnancy. This evaluation should include but is not limited to a serum creatinine, serum urea nitrogen, electrolytes, albumin, cholesterol, complete blood picture urinalysis, and urine culture. In addition, a 24-hour urine collection for volume, creatinine clearance, and protein to monitor for worsening of renal function or the development of preeclampsia should be done.

The goal of drug therapy of hypertension is to reduce fetal morbidity and mortality by preventing severe hypertension and/or preeclampsia and keeping the target diastolic blood pressure around 80-90 mm Hg. For mild hypertension, the central alpha agonist methyldopa and labetalol are safe and effective. For severe hypertension calcium channel blockers like nifedipine, combined alpha and beta blockers like labetalol and hydralazine are used. In hypertensive crisis nitroglycerin infusion is started for acute control of blood pressure. The control of blood pressure should be gradual and monitored closely. Maternal anemia that occurs in pregnant women with chronic renal disease can be managed with oral iron therapy, recombinant erythropoietin, intravenous iron, or blood transfusions. Recombinant erythropoietin when the hematocrit falls below 19% should be strongly considered. The clinician should be cognizant of the fact that erythropoietin may cause or aggravate preexisting hypertension. Erythropoietin is administered in dose of 2000–8000 units subcutaneously once weekly (dose depending upon patient's weight, severity of anemia, and associated symptoms).

Antepartum fetal surveillance should begin at 28 weeks of gestation or earlier, depending on the degree of renal insufficiency, hypertension, fetal growth restriction, and past obstetric outcome (i.e., prior stillborn) (4). Fetal growth is monitored every 3-4 weeks by ultrasonography and fetal surveillance can be modified as per institutional protocol. In the absence of maternal or fetal deterioration, consideration should be given to delivery at or near term and caesarean section should be done for maternal indications.

At present there is paucity of scientific data for specific clinical recommendations regarding renal biopsy during pregnancy. Moreover, specialists prefer to defer the procedure to the postpartum period because of the associated complications, which include gross hematuria, perirenal hematoma, and severe flank pain.

### Special Considerations

#### **Diabetic Nephropathy and Pregnancy**

The occurrence of diabetic nephropathy increases with the duration of diabetes for 10 – 15 years and the prevalence is 5 to 10 %. Women with diabetes, micro

albuminuria, well – preserved renal function, and normal blood pressure have a good prognosis for pregnancy, although they are at increased risk of pre eclampsia and urinary infection. (5,6). Most reproductive age women with overt diabetic nephropathy have preserved renal function and do not seem to have the progression of their disease affected by pregnancy. Women with non-nephrotic range proteinuria in preconception period may develop nephrotic range proteinuria during pregnancy, which is usually reversible. Assessment for vasculopathy is important before pregnancy because nephropathy can increase the perinatal risks. Nephrotic range proteinuria is an indication for thromboprophylaxis with heparin in pregnancy and the puerperium and target blood pressure should be below 140/90 mmHg. Perinatal outcomes are excellent for women who receive care in tertiary institutions.

For women with diabetic nephropathy, adverse perinatal outcomes are more common, and the effect of pregnancy on the course of their disease is less certain (7), so a multicenter surveillance program is needed, in order to study less frequent maternal and neonatal outcomes as well as the long-term effects of pregnancy on the natural course of diabetic renal disease.

#### **Lupus Nephritis(LN) in Pregnancy**

Lupus is an unpredictable illness because of the tendency of the disease to flare. Increased SLE disease activity is expected during pregnancy because of increased levels of estrogen, prolactin, and T-helper cell 2 cytokines. Lupus exacerbation during pregnancy occurs in about 20-30% of pregnant lupus patients.

Renal biopsy sample demonstrating immune complex-mediated glomerulonephritis is optimal criterion for diagnosis of lupus nephritis.

Clinical and laboratory manifestations that meet ACR(American College of Rheumatology) criteria for lupus nephritis - persistent proteinuria more than 0.5 gm per day or greater than 3 plus by dipstick, and/or cellular casts including red blood cells [RBCs], hemoglobin, granular, tubular, or mixed. A spot urine protein/creatinine ratio of 0.5 can be substituted for the 24-hour protein measurement, and "active urinary sediment" (more than 5 RBCs/high-power field ,more than 5 white

blood cells /hpf in the absence of infection, or cellular casts limited to RBC or WBC casts) can be substituted for cellular casts.(8)

### International society for Nephrology/ Renal pathology has classified them as :

Class I	Minimal mesangial LN
Class II	Mesangial proliferative LN
Class III	Focal LN (<50% of glomeruli)
III (A)	Active Lesions
III (A/C)	Active and chronic lesions
III (C)	Chronic lesions
Class IV	Diffuse LN (>=50% glomeruli)
	Diffuse segmental (IV-S) or global (IV-G) LN
IV (A)	Active lesions
IV (A/C)	Active and chronic lesions
IV (C)	Chronic lesions
Class V	Membranous LN
Class VI	Advanced sclerosing LN

Women with lupus nephritis are advised not to conceive unless their disease has been inactive for the preceding 6 months, because active disease is associated with a higher incidence of fetal demise. Disease is considered inactive when the creatinine level is less than 0.7 mg/dl, proteinuria is less than 0.5 gm/dl and on spun urine examination lower than five red blood cells are present per high powered field. Fetal loss occurs in 25% to 50% of women who conceive when their disease is active with a creatinine greater than 1.2 mg/dl. (9)

Pregnancy can be successful in most women with pre-existing Lupus Nephritis, even for those with a severe renal involvement at onset. Renal flares during and after pregnancy are not uncommon but can be predicted by renal status assessed before pregnancy. Patients with anticardiolipin antibodies and lupus anticoagulant are at increased risk of fetal loss and worsening of renal function so they should be screened for anticardiolipin antibodies and lupus anticoagulant activity. Normocomplementaemia and low-dose aspirin

therapy during pregnancy are independent predictors of a favourable fetal outcome. (10).

Adverse fetal outcome including fetal loss, preterm birth, and SGA increases significantly with SLE(Systemic Lupus Erythematosus) flares during pregnancy with preeclampsia/eclampsia , thrombocytopenia, and active SLE serving independent predictors of adverse fetal and maternal outcome. Fetal echo should be done not just for heart block but for structural abnormalities as the structural malformation rate are significantly higher than general population, especially congenital heart disease. (11)

The Task Force Panel recommends in patients with prior LN but no current evidence of systemic or renal disease activity, no nephritis medications are necessary. Patients with mild systemic activity may be treated with Hydroxychloroquine ; this probably reduces activity of SLE during pregnancy. If clinically active nephritis is present, or there is substantial extra renal disease activity, glucocorticoids may be prescribed at doses necessary to control disease activity, and if necessary Azathioprine (AZA) can be added. High-dose glucocorticoid therapy in patients with SLE is associated with a high risk of maternal complications such as hypertension and diabetes mellitus. Mycophenolate mofetil, cyclophosphamide and methotrexate are avoided because they are teratogenic in humans. The dose of AZA should not exceed 2 mg/kg in a pregnant woman. For patients with a persistently active nephritis with documented or suspected class III or IV with crescents, consideration of delivery after 28 weeks for a viable fetus is recommended.

### **Chronic Pyelonephritis**

It is seen in association with recurrent urinary tract infections along with urinary tract abnormalities caused by dilatation and stasis in the urinary tract which is exacerbated in pregnancy. High fluid intake should be encouraged along with screening with urine cultures at least monthly. Prompt treatment with antibiotics is essential and suppressive therapy may be warranted for the duration of pregnancy in some of the cases. (12)

### **Chronic Glomerulonephritis**

Women in reproductive age group can have any forms of chronic glomerulonephritis, including Immunoglobulin

A nephropathy, focal and segmental glomerulosclerosis, membrano proliferative glomerulonephritis, minimal change nephritis, and membranous nephropathy.

It is observed that there is increased incidence of high blood pressure late in gestation but usually no adverse effect if renal function is preserved and hypertension is absent before gestation. Some disagree, believing coagulation changes in pregnancy exacerbate disease, especially immunoglobulin A (IgA) nephropathy, membranoproliferative glomerulonephritis, and Focal Glomerulosclerosis.

Rogov et al (13) studied chronic glomerulonephritis in pregnancy and found high proteinuria (34.6%), progression of hypertension (29.5%), renal function deterioration (15.4%), fetal and neonatal losses (15.4%), fetal underdevelopment (25%), preterm delivery (17.3%), preeclampsia (7.7%), preterm placental detachment (1.9%). Pregnancy does not affect the course of renal disease in patients who have normal renal function at conception. (14) Ultimately baseline renal function and blood pressure are what dictate outcomes.

### **Polycystic Kidney Disease**

Women in reproductive age group with autosomal dominant polycystic kidney disease are frequently asymptomatic, with normal renal function and normal blood pressure and may be unaware of the diagnosis. All patients should undergo pre pregnancy counseling to ensure they are aware that their offspring have a 50% chance of being affected and possible pregnancy complications. Hypertension and urinary tract infection are frequent association. Estrogen is known to cause liver cysts to enlarge and repeated pregnancies may result in symptomatic enlargement. Cerebral aneurysms are also associated with this disease and screening for these aneurysm may be considered before natural labour. (15)

### **Dialysis in Pregnancy**

It is a therapy for end stage renal disease in pregnancy. Management of pregnant patients on dialysis includes several considerations, but the single most important factor influencing fetal outcome is the maternal plasma urea level. (16)

Dialysis should be initiated in pregnancy when serum

creatinine range is 3.5 to 5.0 mg/dL (309 to 442  $\mu$ mol/L) or GFR below 20 mL/min (0.33 mL/s) (17). Fetal outcome is improved with longer more frequent hemodialysis sessions; target is 20 h/wk, aiming for a predialysis urea of 30- 50 mg/dl. These women typically have worsening hypertension, develop premature labor, and have small-for-gestational-age fetuses. Nutritional considerations and proper weight gain are essential for successful pregnancy with recommended weight gain of 0.3 to 0.5 kg/wk in second and third trimesters. Adequate calorie and protein intake is required, 1 g per kilogram body weight per day of protein intake plus an additional 20 g/d has been suggested.

Hypotension should be avoided to lessen chance of fetal hypo perfusion. Peritoneal dialysis can avoid intermittent hypotension and anticoagulation but may increase risk of hypokalemia and peritonitis. Spontaneous abortion rate is about 50% in women on dialysis, but in pregnancies that continue overall fetal survival has been reported as high as 71%. Infant survival is higher when pregnancies are conceived before dialysis is initiated. Oral magnesium supplementation to maintain serum magnesium level at 5 to 7 mg/dl should be used in view of theoretical risk of preterm labour when magnesium levels are low. Babies born to mothers on dialysis may require monitoring for osmotic diuresis in the immediate postpartum period if maternal urea was high at delivery.

### **Pregnancy in Renal Transplant Recipients**

Fertility in women with kidney failure is restored by transplantation. It requires careful planning and is only advisable in women with good kidney function, controlled blood pressure, and general good health. (1,18) Best practice guidelines outline criteria for considering pregnancy in renal transplant recipients, and suggest that those contemplating pregnancy should meet the following:

- Good health and stable renal function for 1 to 2 years after transplantation with no recent acute or ongoing rejection or infections
- Absent or minimal proteinuria (<0.5g/d)
- Normal blood pressure or easily managed hypertension
- No evidence of pelvic/cecal distension on ultrasonography before conception

- Serum creatinine less than 1.5 mg/dl or less; azathioprine 2 mg /kg or less; cyclosporine less than 5 mg/ kg per day.

Immunosuppressive drugs carry risks for the fetus, but the risks of prednisone, azathioprine, cyclosporine, and tacrolimus are surprisingly low. The success rate for pregnancy in kidney transplant recipients is lower than in the general population with 70% to 80% of pregnancies resulting in surviving infants. Prematurity, intrauterine growth restriction, and preeclampsia are all increased. Ten to 15% of women have a temporary or permanent decline in kidney function, particularly if prepregnancy creatinine is high.

### Summary

Women of childbearing age with kidney disease should be made aware of the implications regarding reproductive health and contraception. Pre- pregnancy counseling should be offered which allows discussion of risks and modification of risk factors. Baseline renal function and degree of hypertension are key determinants in deciding the outcome. Women with normal or mildly decreased renal function will usually have successful outcome without adverse effects on long term course of disease .Mothers with renal disorders should be managed at a tertiary centre by a multidisciplinary team. Though these disorders are less common they pose significant maternal and fetal risk, so multicenter studies are required to better identify risks and determine optimal therapeutic strategies.

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## PRE- EXISTING CHRONIC RENAL DISEASE IN PREGNANCY

Name: \_\_\_\_\_ MR No. \_\_\_\_\_

Type of Renal Disease \_\_\_\_\_

### Checklist at Booking

Is hypertension under control?	
Is patient on ACE inhibitors or ARBs?	
What is the level of proteinuria?	
What is the renal function?	
Is there urinary obstruction or stasis?	
What is the underlying disease?	
Is there underlying activity of Lupus?	
Is there a positive anti-phospholipid antibody?	
Is there a history of recurrent UTI?	

### Routine Investigations:

	Date of Investigations					
Hb%						
Urine albumin						
S. Creatinine						
Cr. clearance						
24 hrs Urine Protein						
Urine C/s						
S. Potassium						

### Plan of Monitoring :

	Frequency					
Antenatal checks	Every 2 wks till 32 wks, then wkly					
Investigations	Every 4 weeks					
USG	Dating Scan					
	NT at 11-14 wks, Ut Art Doppler					
	TIFFA at 22 wks					
	Growth scan at 28 weeks					
Betnesol at 28 wk	For fetal lung maturity					

**DELIVER at 34 –36 weeks**

**Date:** \_\_\_\_\_

## SOCIETY OF OBSTETRIC MEDICINE (SOM), INDIA

### Membership Form

Surname : \_\_\_\_\_ First Name : \_\_\_\_\_

Qualification : \_\_\_\_\_ Date of Birth : \_\_\_\_\_

Designation : \_\_\_\_\_

Place of Work Address : \_\_\_\_\_

Hospital \_\_\_\_\_

PHOTO

Type of Hospital: Women's / General / District / Nursing Home Total No. of Beds : \_\_\_\_\_

Year of Passing : MBBS \_\_\_\_\_ Diploma \_\_\_\_\_ MD \_\_\_\_\_ DNB \_\_\_\_\_ Fellowship / Other : \_\_\_\_\_

Medical Council Registration No. : MBBS : \_\_\_\_\_ PG : \_\_\_\_\_

Residence Address : \_\_\_\_\_

Email ID : \_\_\_\_\_

Tel No. : (Res) \_\_\_\_\_ (Off) : \_\_\_\_\_ (Mobile) : \_\_\_\_\_

**Annual Membership Fees** : Rs. 500/- (Rupees Five Hundred only) DD to be drawn in favour of "**Society of Obstetric Medicine, India**" payable at Hyderabad. (Send it with two passport size photos to the address below).

Signature \_\_\_\_\_

#### For Office use only

Registration No. \_\_\_\_\_ Receipt No. \_\_\_\_\_

Received: Cash / DD / Cheque \_\_\_\_\_ Remarks: \_\_\_\_\_



# Obstetric NEWSLETTER Medicine

## SOCIETY OF OBSTETRIC MEDICINE INDIA

### **Aims and Objectives**

- To establish a forum for exchange and interchange of views and for enhancing fellowship among its members and come out with evidence based guidelines for management of medical disorders complicating pregnancy.
- To assist in the establishment of Obstetric Medicine as an important sub-specialty to advance clinical and scientific knowledge of medical illness complicating pregnancy.
- To promote research and training in Obstetric Medicine.
- To foster collaboration with other regional and international societies interested in Obstetric Medicine.
- To carry out all such activities as would contribute to the promotion of Obstetric Medicine.
- To promote formation of patient forums.

### **Inviting Articles for Future Issues**

Theme for next issue (April, 2014) is 'Tuberculosis and Pregnancy'. We invite articles in this subject. Last date for submission is 28<sup>th</sup> February 2014. Please send in your articles (interesting case reports, review articles, original research articles, comments on previous articles) in Word document format to [obsmedindia@gmail.com](mailto:obsmedindia@gmail.com) with a copy to the editor, [drharikishan@gmail.com](mailto:drharikishan@gmail.com).