PRACTICE POINTS - PREGNANCY AND THE KIDNEY

In recognizing renal disease, measurement of kidney function and proteinuria are the early standard bearers of subclinical pathology. With the dramatic hormonal and hemodynamic changes of pregnancy, renal function is altered and these changes must be considered when assessing renal function in pregnancy and in the choice of medications provided through parturition.

ASSESSMENT OF GFR AND PROTEINURIA DURING PREGNANCY

The physiologic increase in GFR during pregnancy normally results in a decrease in concentration of serum creatinine, which falls by an average of 0.4 mg/dl to a pregnancy range of 0.4 to 0.8 mg/dl.Hence, a serum creatinine of 1.0 mg/dl, although normal in a nonpregnant individual, reflects renal impairment in a pregnant woman.

Creatinine-based formulas developed in nonpregnant populations are likely to be inaccurate when applied to pregnant women. Given these issues, 24-h urine collection for creatinine clearance remains the gold standard for GFR estimation in pregnancy.

The quantification of proteinuria in pregnancy is indicated in at least two clinical situations. The first is monitoring of proteinuria in pregnant women with preexisting proteinuric kidney disease. The second important indication for the quantification of proteinuria in pregnancy is for the diagnosis of preeclampsia. It is reasonable to use the urine Protein:Creatinine ratio for the diagnosis of preeclampsia, with 24-h collection under taken when the result is equivocal.

PREGNANCY IN THE SETTING OF CKD

The degree of renal insufficiency, rather than the underlying renal diagnosis, is the primary determinant of outcome. Moderate to severe CKD results in an increased risk for pregnancy complications and neonatal morbidity: More than 70% of women who become pregnant with a serum creatinine>2.5 mg/dl will experience preterm delivery, and >40% develop preeclampsia.

PREGNANCY AFTER KIDNEY TRANSPLANTATION

Fertility rates increase dramatically after transplantation in women with end-stage kidney disease; therefore, pregnancy is common among young female transplant recipients. Most evidence suggests that pregnancy after transplanta- tion does not increase risk for graft loss. so long as renal function is good (creatinine <1.5 mg/dl with no proteinuria) and the patient is on a stable immunosuppressive regimen. In this situation, rejection rates are similar to the general transplant population, and there does not seem to be an increased risk for birth defects. Neonatal outcomes in pregnancies among renal transplantation patients are generally good. Calcineurin inhibitors, steroids, and azathioprine are the mainstays of safe immunosuppressive therapy in pregnant transplant recipients. Ace inhibitors have to be stopped if pregnancy is planned.

CURRENT PATHOGENESIS OF PREECLAMPSIA

Placental dysfunction, triggered by poorly understood mechanisms—

including genetic, immuno- logic, and environmental—plays an early and primary role in the development of preeclampsia. The damaged placenta in turn secretes the antiangiogenic factors, sFlt1 and sEng, into the maternal circulation. These factors lead to impaired VEGF/PlGF and TGF-□signaling, resulting in systemic endothelial cell dysfunction mediated by a variety of factors, Endothelial dysfunction, in turn, results in the systemic manifestations of preeclampsia The role of aspirin, calcium and folic acid , l arginine antioxidants and anti angiogenic factors are being tried in the prevention and treatment of pre eclampsia.

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