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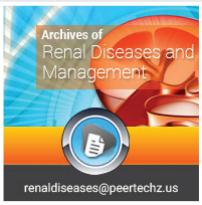

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Short Communication

Estimation of Glomerular Filtration Rate (GFR) in pregnant women

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Pregnancy induces marked changes in the cardiovascular and plasma volume regulatory systems. By the end of the first trimester of gestation, the kidneys show marked glomerular hyperfiltration, resulting in a decrease in the patient's serum creatinine with advancing gestational age [1]. As a consequence, the Glomerular Filtration Rate (GFR) will increase by approximately 50% - 60% above the pre-pregnancy value [1–3]. During pregnancy, there is a state of vasodilatation and volume expansion, hemodynamic changes in which several systems are involved, among them is the renin-angiotensin system and maternal hormones. Progesterone increases renal plasma flow and glomerular filtration, although it alone does not explain the magnitude of the increase in glomerular filtration rate [4]. The hormone relaxin appears to be involved in the mechanism of glomerular hyperfiltration and increased renal blood flow during pregnancy. This hormone is secreted by the corpus luteum, placenta and decidua in response to high doses of chorionic gonadotropin. Relaxin causes generalised vasodilatation and decreased resistance of afferent endothelin in the renal circulation. Following its action, renal blood flow increases [5]. In a recent study of renal function in 243,534 pregnant women, the mean creatinine concentration was 0.68 mg/dl before pregnancy, decreasing rapidly at 4 weeks gestation to a nadir of 0.53 mg/dl between 16 and 32 weeks gestation. After 32 weeks gestation, there was a steady increase in serum creatinine levels, reaching a peak of 0.72 mg/dl within a few weeks postpartum, with a gradual return to mean pregestational levels at 18 weeks postpartum[6]. Creatinine-based equations used to estimate glomerular filtration may therefore misclassify renal function during pregnancy, as they rely on a steady state of creatinine equilibrium [3,6], which does not correspond to the dynamic nature of changes in renal function during gestation and postpartum.

Renal impairment can occur during pregnancy in women without pre-existing renal problems, especially in the context of pre-eclampsia [3]. Renal function decreases in hypertensive pregnancy [7]. These cases have an increased risk of developing complications during pregnancy or even neonatal complications. Monitoring renal function in pregnant women is crucial to reducing the rate of adverse events, both in women with pre-existing kidney disease and in women who develop kidney disease during pregnancy [8]. Several studies report that cystatin C is a better marker than serum creatinine for pre-eclampsia, as well as a reliable marker of the glomerular inflammation found in pre-eclampsia [9]. Furthermore, the increase in cystatin C levels in term pregnancies is not due to uteroplacental production of cystatin C [10] considering that cystatin C increases in the second trimester of gestation as a consequence of other gestational factors [11]. Therefore, the increase in plasma cystatin C concentration in women with pre-eclampsia and the decrease in GFR in the antepartum period is probably due to a true decrease in GFR [10].

If we explore the behavior of glomerular filtration rate estimation by equations in the pregnant state we can conclude:

- The MDRD equation significantly underestimates the estimation of the glomerular filtration rate in both healthy pregnant women and pregnant women with pre-eclampsia [4]. It is not sensitive enough to be used as a screening test for kidney disease in this population.

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Compared to inulin clearance, MDRD underestimates GFR in pregnancy by up to 41 ml/min per 1.73 m2 [3] so clinical guidelines advise against its use in pregnancy [8].

- Estimation of glomerular filtration rate in pregnant women with pre-eclampsia does not provide a reliable result of renal function. A new equation, the preeclampsia GFR (PGFR) formula, has been developed for women with pre-eclampsia at 24 weeks gestation and requires further study to assess whether it is more accurate in assessing GFR in these clinical circumstances than known equations (Cockcroft Gault, MDRD, or CKD-EPI) [12].

- Estimated glomerular filtration rate (eGFR) derived from CKD-EPI compared to GFR assessment by inulin clearance underestimates formal GFR by up to 20% in pregnancy [13]. When comparing glomerular filtration rate estimation in pregnant women using CKD-EPI with the measurement of glomerular filtration rate in 24-hour urine, CKD-EPI had a bias of 14.2 ml/min/1.73 m^2 [14].

- A recently formulated estimation equation, the Nanra equation, seems a priori to guarantee an adequate estimation of glomerular filtration rate for pregnant women, given that in its development it ignores body weight, which is highly variable during pregnancy and introduces the variable height. However, studies comparing the estimation of glomerular filtration rate in pregnant women using the Nanra equation and glomerular filtration rate obtained by creatinine clearance in 24-hour urine did not show appropriate concordance [14].

Reasonable doubt in these studies is raised by the fact of using creatinine clearance in 24-hour urine as a comparator to verify the behavior of the filtration equation. This system alone offers enormous intraindividual and interindividual variability.

Based on existing evidence, the Clinical Practice Guideline on Renal Disease in Pregnancy recommends that: "Renal function in pregnancy should be assessed by serum creatinine levels as estimated GFR (eGFR) is not valid for use in pregnancy (grade of evidence 1C)" [11]. This recommendation is reinforced by the fact that adverse events during pregnancy can be predicted based on creatinine levels stratified by gestational age [15].

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