Renal Failure in Pregnancy

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KEYWORDS

• Renal failure • Pregnancy • Preeclampsia • HELLP • Thrombotic microangiopathies • Acute fatty liver of pregnancy • Dialysis

KEY POINTS

- Renal failure in pregnancy is a rare but severe complication that can have significant long-term effects on both mother and fetus.
- Evaluation of the pregnant patient must take into consideration normal physiologic changes of pregnancy that increase the complexity of diagnosing renal failure.
- The etiology of renal failure in the pregnant patient is divided into prerenal, intrarenal, and postrenal causes; treatment focuses on the underlying cause.
- Supportive measures such as fluid resuscitation are the basis of treatment for renal failure in pregnancy, with delivery usually being the ultimate goal.
- When other measures fail dialysis may be required, with the goal of prolonging pregnancy until delivery becomes feasible, and providing an environment suitable for fetal growth.

INTRODUCTION

Renal failure is a potentially devastating complication during pregnancy that can affect both mother and fetus. It may be related to preexisting renal disease, or may develop as a new entity during pregnancy. Preexisting chronic renal disease occurs in about 4% of parturients. These women have a 43% increased risk of pregnancy-related renal dysfunction, and 10% of patients will develop a rapid deterioration of renal function.[1,2] Acute renal failure (ARF) presents an important clinical challenge and, although rare, it can be associated with significant morbidity and mortality. In the nonpregnant patient, ARF has been defined by various changes including serum creatinine increased by 0.5 mg/dL or greater over baseline, or a greater than 50% increase over baseline, or a 50% reduction in creatinine clearance, or renal dysfunction requiring dialysis.[3] Oliguria is often seen with ARF in pregnant patients, defined as urine output of less than 0.5 mL/kg/h. Although this definition has been standardized for the nonpregnant patient by the Risk–Injury–Failure–Loss–End (RIFLE) classification

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system,[4] no such standard definition exists for the parturient, although Mantel[5] has used a clinical definition of oliguria of less than 400 mL per 24 hours not responsive to therapy, or an acute increase in serum urea to greater than 15 mmol/L or creatinine of greater than 400 mol/L. The incidence of ARF has decreased from 1/3000 to 1/15,000 to 1/20,000 since the 1960s, mainly owing to improved prenatal care and decreases in septic complications, such as abortions. Despite the decrease in incidence, overall mortality rates have remained in the 0% to 30% range, and the long-term prognosis has also remained consistent, with full recovery rates of 60% to 90% after episodes of ARF.[2] To effectively evaluate and treat the pregnant patient with renal failure, the underlying etiology must be identified correctly.

PATIENT EVALUATION OVERVIEW

Evaluating renal failure in the parturient can be a complex task, especially given the lack of a commonly accepted definition of the condition. Many different factors must be considered, including the normal physiologic changes of pregnancy and the variety of conditions that can produce renal failure. Determination of the etiology is extremely important, because the treatment will depend on the underlying cause.

The normal physiologic changes of pregnancy add a layer of complexity to evaluating renal failure in the parturient compared with the nonpregnant patient. During pregnancy, kidneys can normally undergo a 30% increase in size, and hydronephrosis may cause an even further enlargement. The glomerular filtration rate (GFR) will increase to 30% to 50% more than in nonpregnant women, primarily owing to a reduced average oncotic pressure and an increase in ultrafiltration, and renal plasma flow will initially increase beyond and then decrease below the GFR, leading to increased filtration toward the end of pregnancy.[6] The normally occurring changes in renal physiology during pregnancy lead to increased an intravascular volume owing to hormonal changes, a normal increase in protein excretion of up to 300 mg/24 h, an increase in creatinine clearance to 120 to 160 mL/min, and a decrease in serum creatinine to 0.4 to 0.7 mg/dL.[1,7]

Because the increased GFR and hemodilution from increasing intravascular volume during pregnancy causes an expected decrease in serum creatinine, there is an intrinsic inaccuracy in applying commonly used measures to evaluate renal disease in the case of pregnancy. The Modification of Diet in Renal Disease formula that is frequently used to estimate GFR in patients with chronic kidney disease underestimates the GFR when the GFR is greater than 60 mL/min/m,[2] and the Cockcroft–Gault formula for estimating GFR is weight based, and tends to overestimate GFR in the pregnant patient owing to increased body weight not being from increased muscle mass.[8] For these reasons, the gold standard for estimating GFR in the pregnant patient is 24-hour urine collection for creatinine clearance. It is also useful to establish whether a decrease in the GFR is secondary to renal vasoconstriction or systemic hypoperfusion with intact tubular function or established ARF. A fractional excretion of filtered sodium (FE_{Na} = [(urine sodium × plasma creatinine)/(plasma sodium × urine creatinine)]) of less then 1% is indicative of preserved tubular sodium reabsorbtion and intact tubular function.

Although a renal biopsy may be useful in nonpregnant patients, it tends to be less useful in the evaluation of pregnant patients owing to there being a higher chance of already knowing the underlying etiology. The major role of biopsy is for the identification of renal failure not owing to preeclampsia, to find a therapy other than delivery.[2]