

Acute Renal Damage in Kidney Transplant Patients during Pregnancy

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ABOUT THE STUDY

In 1958, a lady with a kidney transplant became pregnant for the first time. Subsequent pregnancies in women following kidney transplantation demonstrated that pregnancies with satisfactory maternal and foetal outcomes were attainable. Women with chronic renal illness have decreased fertility. Regular menstrual and ovulatory cycles can resume as soon as 3 weeks following kidney transplantation and the hypothalamic-pituitary-gonadal axis is returned to normal by 6 months. KDIGO guidelines urge avoiding pregnancy until at least the first year after transplant to allow kidney function to stabilize for the best outcome. However, given the possibility of foetal, maternal, and allograft problems impacting outcomes, post-transplant pregnancy is not a "zero risk" option.

While preeclampsia, allograft rejection, premature deliveries, and stillbirths are all known as potential pregnancy problems in women who have had kidney transplants, Acute Kidney Injury (AKI) is rarely discussed in the literature. As a result, there is no definitive evidence on the prevalence of AKI among pregnant kidney transplant patients.

Because to advanced maternal age, increasing comorbidities, lifestyle disorders such as hypertension, diabetes, and obesity, and greater detection rates, the prevalence of pregnancy related AKI has nearly tripled in industrialized nations. In both kidney transplant patients and the general population, there is significant variation in the classification and diagnosis of pregnancy related AKI. There is a considerable knowledge gap due to the scarcity of data on the various etiologies and consequences of AKI during pregnancy in women receiving kidney transplants.

Effects of pregnancy on kidney allograft function

GFR increases by 40%-60% in a normal pregnancy due to hyperfiltration, vasodilation, and an increase in effective plasma flow, which enhances clearance of blood urea nitrogen and creatinine. During pregnancy, the serum creatinine concentration normally falls below. Blood urea nitrogen levels fall below 12 mg/dl. Glomerular hyperfiltration causes physiologic proteinuria during pregnancy. The kidney allograft responds to the physiological changes that occur during pregnancy. Creatinine clearance increases by approximately 30% during the first trimester, slightly decreases during the second trimester, and returns to prepregnancy levels by the third trimester. The absence of a drop in serum creatinine during early pregnancy indicates a bad prognosis, and doctors should carefully evaluate kidney transplant patients whose creatinine does not decline with predicted physiologic changes of pregnancy. It has been shown that among women with chronic kidney disease stages 3-5, a 10% drop in blood creatinine during pregnancy (together with chronic hypertension and proteinuria) was related with unfavourable pregnancy and renal outcomes (preterm delivery, low birthweight and loss of maternal kidney function). Furthermore, an increase in GFR increases pregnant proteinuria, and women with kidney transplants had greater 24 hour urine protein excretion than healthy women. Protein excretion in pregnant kidney transplant patients may rise up to thrice during the third trimester, hitting 500 mg compared to 200 mg in healthy pregnant women, before returning to normal levels three months after delivery. An unexpected rise in proteinuria from baseline during pregnancy in women who have had a kidney transplant should prompt a search for other causes of AKI, such as preeclampsia and urinary tract infection.

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Received: 29-Aug-2022, Manuscript No. RSSD-22-19646; **Editor assigned:** 02-Sep-2022, PreQC No. RSSD-22-19646 (PQ); **Reviewed:** 16-Sep-2022, QC No. RSSD-22-19646; **Revised:** 23-Sep-2022, Manuscript No. RSSD-22-19646 (R); **Published:** 30-Sep-2022, DOI: 10.35248/2161-038X.22.11.332.

Citation: Zara S (2022) Acute Renal Damage in Kidney Transplant Patients during Pregnancy. *Reprod Syst Sex Disord*. 11:332.

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