Dear Sir,

Some diseases of the kidney are encountered in young women of childbearing age, and thus they may complicate pregnancy. In some cases, the diseases first appear during pregnancy, and in others renal function deteriorates rapidly and leads to maternal and fetal death [1]. Rapidly progressive glomerulonephritis (RPGN) is a rare cause of renal failure in association with pregnancy [1]. We report a patient with normal renal function and blood pressure and without proteinuria before pregnancy, whose renal function progressively deteriorated during pregnancy due to RPGN followed by spontaneous abortion.

A 26-year-old woman who was now gravida 1, para 1 presented at 20 weeks' gestation with malaise, cola-colored urine, decreased urine output and bilateral ankle edema. During the last 2 weeks, her edema increased and mounted to her legs. She had no history of renal disease. Her previous admission at 12 weeks' gestation was not complicated by edema, hypertension or proteinuria. On her second admission at 20 weeks of gestation, blood pressure was 160/100 mm Hg. Physical examination was unremarkable except for bilateral ankle and leg edema. The results of urinalysis were specific gravity 1.016, pH 5.4+ protein, and urine sediment examination revealed red cell casts and fat bodies. She had 6.0 g protein in a 24-hour urine collection. Complete blood count results were white blood cells 12,400/mm3, hemoglobin 10.2 g/dl and platelet count 256,000. Serum biochemistry results were as follows: BUN, 136 mg/dl; creatinine, 12.6 mg/dl; Na, 138 mEq/l; K, 6.2 mEq/l; Cl, 96 mEq/l. Total protein and albumin were 5.6 and 2.4 g/dl, respectively. Serum transaminases were within normal limits. The serum antinuclear antibody was negative. The serum anti-glomerular-basement-membrane and anti-neutrophilic-cytoplasm antibodies were also negative. The complement levels were within normal limits. Serological tests were negative for hepatitis B and hepatitis C. Abdominal ultrasonography showed mildly enlarged kidneys. Her condition worsened during the last 2 weeks and her pregnancy was complicated by spontaneous abortion. A renal biopsy was performed, and 'crescentic glomerulonephritis without immune deposit' was diagnosed. The patient was treated with intravenous pulse cyclophosphamide (500 mg) and oral prednisolone (1 mg/kg/day), but the patient’s renal
function did not recover and she underwent a hemodialysis program within 4 weeks after her admission.

The most common cause of proteinuria, hypertension and edema in pregnancy is preeclampsia and in one large series, one third of the preeclamptic patients with preexisting renal disease developed nephrotic-range proteinuria during gestation [2]. However, irreversible renal functional loss occurs rarely during pregnancy in a preeclamptic patient [2, 3]. In this patient, irreversible renal failure and red cell casts in the urine sediment suggested that she had probably an induced pregnancy glomerular disease, and renal biopsy showed RPGN without immune deposit. Glomerulonephritis rarely develops acutely during pregnancy, in which it may mimic preeclampsia [1]. Most of the cases of acute glomerulonephritis in pregnancy reported previously in the literature represent in fact exacerbations of the chronic disease. Besides, acute glomerulonephritis may result from any of several causes, including infectious diseases, multisystem diseases or primary disorders unique to the glomerulus during pregnancy. Rarely, there may also be a pregnancy-induced glomerular disease. Rarely, some cases of acute glomerulonephritis in pregnancy never completely recover, and RPGN leads to end-stage renal failure within weeks to months. RPGN is a rare cause of renal insufficiency in association with pregnancy [1]. On the other hand, whatever the cause, acute glomerulonephritis and RPGN have a profound effect on pregnancy outcome [4]. Although, in 3 previously reported cases, the patients with RPGN due to Goodpasture’s syndrome carried a successful pregnancy to term, we think RPGN complicates pregnancy and may lead to maternal and/or fetal death [5-7]. So, the relationship and mechanism by which pregnancy causes any form of glomerulonephritis has yet to be elucidated and needs further clinical investigations.

References