Correspondence

Emphysematous pyelonephritis with acute kidney injury in the postpartum period

Emphysematous pyelonephritis (EPN) causing acute kidney injury (AKI) in the postpartum period is rare.[1-3] We present a patient with unilateral EPN and AKI in the early postpartum period. A 37-year-old, para$_3$ live$_3$, presented in the 1st week of puerperium with high-grade fever, chills and rigors and left loin pain for 2 days. She had no history of dysuria, cloudy urine, haematuria, oliguria, pedal oedema or foul-smelling discharge from vagina. She had no history of pre-eclampsia. She delivered a live full-term male baby by normal vaginal delivery at home. On examination, pulse rate was 124 bpm, blood pressure was 80/60 mm of Hg, temperature was 102°F and pedal oedema was also present. Left lumbar and hypochondrium tenderness and guarding were evident. Investigations revealed serum creatinine of 2.7 mg/dL, haemoglobin of 9.1 g/dL, total leucocyte count of 138,000 per mm^3 and platelet count of 94,000 per mm^3; ultrasound abdomen revealed the right kidney measuring 10.6 cm × 4.8 cm and left kidney measuring 11.2 cm × 5.2 cm and a hypoechoic lesion with air foci of size 4.6 cm × 2.7 cm also noted in upper pole of the left kidney. Urine and blood cultures grew no organisms. Vaginal swab grew *Enterococcus feacalis*. Computed tomography (CT) showed Type I[4] and Class 2[5] EPN [Figure 1]. She was treated with injection piperacillin-tazobactam and injection metronidazole. Under CT guidance, percutaneous drainage was established. Pus drained through percutaneous nephrostomy was sterile. She did not require any renal replacement therapy. Fever and renal function improved.

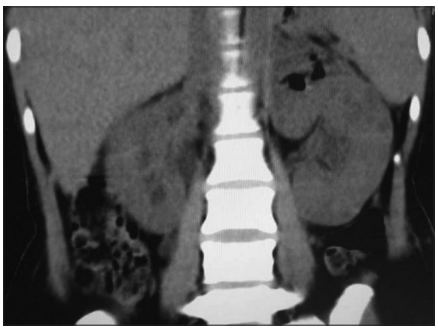

Figure 1: Computed tomographic scan (coronal view) showing enlarged left kidney with pockets of air in the upper pole

While asymptomatic bacteriuria in non-pregnant women is generally benign, pregnant women with bacteriuria have an increased susceptibility to pyelonephritis. The dilatation of renal pelvis and ureters, displacement of bladder superiorly and anteriorly, mechanical compression by the enlarging uterus, smooth muscle relaxation induced by progesterone, pregnancy-induced glycosuria and aminoaciduria and alterations in the urinary pH and osmolality have been cited as the facilitators for bacterial growth.[6] One of the largest published experiences of EPN was of 48 patients, in whom diabetes mellitus was present in 96% and urinary tract obstruction in 22%.[5] The functional ureteric changes in pregnancy might have been a risk factor in our patient.

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Conflicts of interest

There are no conflicts of interest.

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