Case Report

A rare cause of pregnancy-related acute kidney injury-infective endocarditis-associated infection-related glomerulonephritis

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ABSTRACT

Pregnancy-related acute kidney injury (PRAKI) commonly occurs due to septic abortion and accounts for 15–25% of acute kidney injury (AKI). Glomerulonephritis is a rare cause of PRAKI. The index case was a 20-year-old primigravida who presented in the last trimester of pregnancy with infective endocarditis-associated glomerulonephritis, also complicated by splenic infarct, AKI, and intrauterine fetal death. Renal biopsy revealed crescentic glomerulonephritis and she improved on antibiotic and steroid therapy.

Key words: Culture negative, Endocarditis, Endocarditis-associated glomerulonephritis, Normal complements, Pregnancy-related acute kidney injury

In developing countries, pregnancy-related complications account for 15–25% of all cases of acute kidney injury (AKI) and septic abortion accounts for the most cases of pregnancy-related AKI (PRAKI) [1-3]. Although commonly, PRAKI occurs commonly due to preeclampsia and puerperal sepsis, etiologies seen in non-pregnant state can also be encountered in the setting of AKI during pregnancy (Table 1).

Herein, we present a case of infective endocarditis-associated glomerulonephritis causing PRAKI. We are reporting this case owing to the rarity of infective endocarditis-associated glomerulonephritis (IEAGN) as a cause of pregnancy-related AKI.

CASE REPORT

A 20-year-old primigravida, booked pregnancy with uneventful antenatal history from Eastern Uttar Pradesh belonging to low socioeconomic status, presented to us with a history of intrauterine death 2 days before the admission at 38 weeks of gestation. The patient complained of high-grade fever for 20 days before delivery, followed by smoky urine, oliguria, and breathlessness. The patient also complained of persistent pain in the left upper quadrant which was sharp and localized just below the costal margin for 10 days. At the time of referral to the local maternity center, the patient also complained of leaking per vaginum for 1 day and absent fetal heart sounds. Following vaginal delivery, she was referred to the nephrology department in view of renal dysfunction and shortness of breath.

At a presentation to our center, the patient was conscious, irritable, and dyspneic with a blood pressure of 118/78 mmHg, pulse rate of 118/min, SpO_2 of 92%, and the patient was febrile to touch. Pallor and pitting edema were present. In cardiovascular system examination, soft S1 was present along with the pan systolic murmur of mitral regurgitation radiating to the axilla and back.

On investigation, at the time of admission, the patient was found to have advanced renal failure at baseline with serum creatinine 7.4 mg/dL, potassium of 5.8 mmol/L, and bicarbonate of 12 mmol/L, with routine urinalysis showing 4+ proteinuria with plenty of RBCs mostly dysmorphic. The 24-h urinary protein was 2.8 g/day (with a urinary volume of 300 mL). Her total leukocyte count (TLC) was 32190 cells/mm^3 with increased serum procalcitonin of 34.9. Blood cultures were sent which came out to be sterile repeatedly.

The immunological profile and other investigations done at the time of admission in view of Rapidly progressive renal failure (RPRF) revealed normal complements and negative antinuclear antibody and other serologic markers. The chest radiograph was suggestive of pulmonary edema. 2D echocardiography revealed a large mobile mass of 15 mm × 9 mm anterior mitral leaflet with moderate mitral regurgitation and global hypokinesia of the left ventricle with an ejection fraction of 30%. Ultrasound abdomen showed splenomegaly with a wedge-shaped (3.9 cm × 2.3 cm) splenic infarct.

She was initially managed for control of hyperkalemia, volume overload, and infection with empirical antibiotics and received

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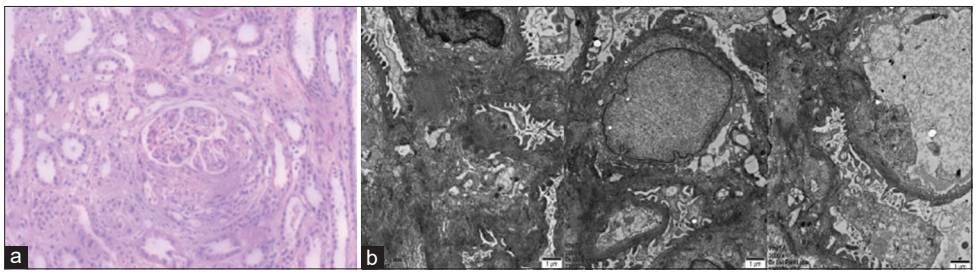

Figure 1: Histopathology of renal biopsy tissue (a) light microscopy H&E stain-cellular crescent and (b) electron microscopy: electron dense deposits (conventional) in mesangial, paramesangial and subendothelial regions of glomerular capillaries. Occasional large subepithelial "hump" like deposits are identified

Table 1: Causes of pregnancy-related acute kidney injury

Pregnancy-related complications	Septic abortion, abruption placentae, placenta previa, uterine hemorrhage, and puerperal sepsis.
Disorders specific to pregnancy	Preeclampsia/eclampsia, HELLP syndrome, acute fatty liver of pregnancy, atypical HUS, TTP, and hyperemesis gravidarum
Causes not related to pregnancy	Acute post infectious glomerulonephritis, lupus nephritis, acute pyelonephritis, dehydration, and urinary tract calculi.

13 sessions of hemodialysis over a period of 6 weeks during the hospital stay. The renal biopsy was sought in view of persistent renal dysfunction and activity in the urine despite improvement in markers of infection.

In renal biopsy, we found all 9 glomeruli with cellular crescents without tuft necrosis on light microscopy with Interstitial fibrosis and tubular atrophy (IFTA) <10%. In direct immunoflourescence (DIF), we had C3 2+ mesangial granular deposits and the rest staining was negative (Fig. 1a). In electron microscopy (EM), electron-dense deposits (conventional) in mesangial, para mesangial, and subendothelial regions of glomerular capillaries. Occasional large subepithelial "hump" like deposits are identified. Impression on renal biopsy was crescentic glomerulonephritis probably infection-related glomerulonephritis (IRGN) (Fig. 1b).

The patient was administered intravenous pulse methylprednisolone for 3 days followed by oral steroids, in view of cellular crescents and no renal recovery. Gradually, urine cleared of its cola color over the next 3 weeks; urine routine showed protein-nil; RBC's 2, 3, and 24-h urinary protein of 100 mg/day with urine output increased to 2.5 L/day. After 10 weeks of follow-up, serum creatinine improved to 1.34 mg/dL. The size of the vegetation has reduced to 8 mm × 4 mm, at the last follow-up.

DISCUSSION

According to Nasr *et al*., the diagnostic criteria for IRGN are met when at least three of the following are present: (a) presence of infectious disease before or at the onset of glomerulonephritis, (b) hypocomplementemia, endocapillary proliferative, and exudative glomerulonephritis on optical microscopy of renal biopsy tissue, (c) C3-dominant deposits on immunofluorescence, and (d) the presence of hump-like subepithelial deposits on EM [4]. Our patient met three out of the above five criteria. Low complement levels are found in only 50% of cases. The most common presentation of IEAGN is acute renal failure while rapidly progressive glomerulonephritis (RPGN) presentation is rare. The pathogenesis of various forms of IRGN is largely driven by the deposition of complement with or without associated immunoglobulins followed by the subsequent immune/inflammatory response.

IRGN in pregnancy is rare, with a small number of case reports published as early as 1980 by Singson *et al*. [5]. The rarity of cases in pregnancy is possibly due to the young age group seen in the majority of cases of IRGN but also begs the question of whether autoimmune mechanisms specific to IRGN are dampened as other autoimmune diseases [6]. To the best of our knowledge, there were no previously reported cases of PRAKI-secondary to IEAGN with normal complement levels and negative blood cultures.

When cultures are negative, the clinician should decide whether to repeat the blood culture or start empiric therapy with a broad-spectrum antibiotic, while taking into account the patient's general condition. As in our case, cultures were repeatedly negative so we continued with empirical antibiotics for 6 weeks and sent cultures repeatedly. In empiric therapy, antibiotics are selected that provide coverage against the most common causative bacterial species, while paying attention to other aspects such as the onset pattern and patient characteristics. The patients usually require long-term antibiotic therapy, irrespective of causative species, and valve replacement may also be necessary [7,8]. In IEAGN with necrotizing lesions or crescent formation, immunosuppression does not improve renal prognosis, rather it is associated with increased mortality. Steroids may be considered in cases of protracted glomerulonephritis [9]. We started the patient on empiric steroids in view of cellular crescents and non-improving renal function, while under the cover of antibiotics.

CONCLUSION

Infective endocarditis-associated crescentic glomerulonephritis in late pregnancy with normal complement levels and RPGN presentation is a relatively rare presentation. In our patient, as repeated cultures were negative, we continued with empirical antibiotics and pulse methylprednisolone followed by oral prednisolone in view of crescentic glomerulonephritis and protracted renal failure. It is difficult to conclude whether the

improvement in renal function in the present case was due to the control of infection or the administration of steroids.

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