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A case of acute cortical necrosis due to hyperemesis gravidarum

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Abstract

Acute cortical necrosis (ACN) is a rare and unique cause of acute kidney injury (AKI). Though ACN is seen in multiple settings, it is most commonly described in the setting of pregnancy. Usual causes in pregnancy are placental abruption and septic abortion. We report a case of ACN in a pregnant patient who presented with hyperemesis gravidarum. Hyperemesis causing such severe form of AKI has not been reported in literature.

Case Presentation

A 22-year-old Asian woman second gravida 12 weeks of gestation presented with 3 days history of multiple episodes of vomiting and decreased urine output. There was no history of abdominal pain, fever or diarrhea. Her first pregnancy was uneventful. She had received volume expansion before referral to our centre. On admission patient was anuric and had facial puffiness. On evaluation her blood pressure was 130/80 mm Hg. Blood investigations showed hemoglobin of 9 g/dl, platelet count of 180×10^3 µl and serum creatinine was 6.4 mg/dl. Liver function tests and coagulation profile were normal. Blood gases showed metabolic acidosis. Her serum lipase and lipase levels were normal. Urine examination showed 1+ proteinuria and 5-10 pus cells/hpf. Urine culture showed no growth. She was diagnosed as a case of hyperemesis gravidarum with acute kidney injury (AKI). She was scheduled for hemodialysis treatment. She had spontaneous abortion three days later. Patient continued to be anuric and finally became dialysis dependent.

Ultrasound examination showed normal sized kidneys. She underwent renal biopsy in third weeks of renal failure. It showed acute tubular necrosis along with necrosis of glomeruli (ghost glomeruli). This was suggestive of ACN (Figure 1).

Core tip

Acute cortical necrosis (CAN) is a rare and unique cause of acute kidney injury (AKI). Though ACN is seen in multiple settings, it is most commonly described in the setting of pregnancy. Usual causes in pregnancy are placental abruption and septic abortion. We report a case of ACN in a pregnant patient who presented with hyperemesis gravidarum, which is a rare presentation of this disease.

Discussion

Multiple episodes of vomiting can cause volume depletion and pre renal AKI. If severe vomiting occurs or if not treated early, patient may develop acute tubular necrosis (ATN). Our patient developed the most severe form of AKI named as "ACN." ACN is a rare and unique form of AKI. This form of AKI is usually described in the setting of septic abortion in early pregnancy. In late pregnancy, ACN is described in case of placental abruption, massive post-partum hemorrhage, severe pre-eclampsia/eclampsia and prolonged intrauterine death. ACN caused by hyperemesis gravidarum has not been described in literature.

ACN accounts for 2% of all causes of AKI in developed countries (1). It is more common in the developing countries. In a study from India the incidence of ACN was 3.12% of all cases of acute renal failure of diverse etiology. Most common cause of ACN was obstetric events (56.2%). The incidence of ACN in obstetric ARF was 15.2 % (2).

Pathogenesis of ACN remains unknown. It is proposed that both primary disseminated intravascular coagulation (DIC) and severe renal ischemia (causing endothelial damage and secondary fibrin deposition) are

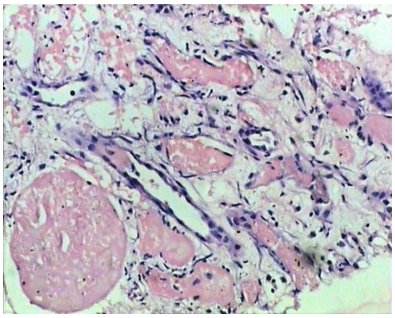

Figure 1. High power view of kidney biopsy showing acute tubular necrosis and necrosis of glomeruli. (Hematoxylin and eosin stain). These features are suggestive of acute tubular necrosis (ATN) (400×).

initiating event in this disorder. When endothelial injury is mild, the local release of nitric oxide normally minimizes the degree of thrombus formation by diminishing platelet aggregation. If the endothelial injury is severe enough to impair nitric oxide release, then the tendency to thrombosis will be increased. Thus severe endothelial dysfunction may cause ACN (3,4).

ACN can be complete or patchy. Calcification in cortical necrosis is seen in cases where partial recovery occurs and patient survives for weeks/months (5).

Renal biopsy is essential to establish the diagnosis. Histopathology shows acute tubular necrosis with associated glomerular necrosis (ghost glomeruli) (6).

Ultrasonography may show hypoechoic outer rim of cortex (7). Computerized tomogram may show a low-attenuation, circumferential band adjacent to the renal capsule is seen that corresponds to the histologic zone of cortical necrosis (5-7). Some authors suggest that computed tomography (CT) findings are classical and may obviate need for renal biopsy (5).

Prognosis of this condition remains poor and almost all patients require dialysis. One study found that partial recovery of renal function occurred in 19.2%. Twenty-eight percent patients progressed to end-stage renal disease (ESRD) (2).

To the best of our knowledge we did not find any published case reports or literature about hyperemesis gravidarum causing ACN. In the present case ACN occurred as a complication of hyperemesis gravidarum.

Authors' contribution

The authors wrote the manuscript equally.

Conflicts of interest

The authors declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, misconduct, data fabrication, falsification, double publication or submission, redundancy) have been completely observed by the authors.

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References

1. Grünfeld JP, Ganeval D, Bournérias F. Acute renal failure in pregnancy. Kidney Int. 1980;18:179-91.
2. Prakash J, Vohra R, Wani IA, Murthy AS, Srivastva PK, Tripathi K, et al. Decreasing incidence of renal cortical necrosis in patients with acute renal failure in developing countries: a single-centre experience of 22 years from Eastern India. Nephrol Dial Transplant. 2007;22:1213-7.
3. Jha V, Sakhuja V. Postpartum renal cortical necrosis. Nephrol Dial Transplant. 2005;20:1010.
4. Shultz PJ, Raij L. Endogenously synthesized nitric oxide prevents endotoxin-induced glomerular thrombosis. J Clin Invest 1992; 90:1718.
5. Fogo AB. Approach to renal biopsy. Am J Kidney Dis 2003; 42:826-36.
6. Sefczek RJ, Beckman I, LupetinAR , Dash N. Sonography of acute renal cortical necrosis. AJR 1984; 142:553-4.
7. Goergen TG, Undstrom AR, Tan H, Lilley JJ. CT appearance of acute renal cortical necrosis. AJR Am J Roentgenol. 1981;137:176-7.