Acute gastroenteritis induced renal cortical necrosis: A rare cause of acute kidney injury

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Abstract

Renal cortical necrosis is a rare cause of acute renal failure, occurs due to ischemic necrosis of the renal cortex. Acute renal failure as a result of renal cortical necrosis cannot be distinguished from other causes of acute renal failure like acute tubular necrosis and the renal biopsy is the only way to make conclusive diagnosis. Acute renal cortical necrosis leads to rapid decline in renal function which is of irreversible in nature and very soon, the patient becomes dialysis dependent if proper intervention not done at the earlier phase of illness. Here, we are presenting such a case of acute renal cortical necrosis, resistant to treatment and ultimately ended into permanent renal loss.

Keywords: Renal Cortical Necrosis, Acute Kidney Injury, Acute Gastroenteritis, Ischemic Glomerular Necrosis.

Introduction

Renal cortical necrosis was first described in 1983 by Friedlander. Acute renal cortical necrosis is an uncommon cause of acute kidney injury. It accounts for only 2% of all causes of acute kidney injury in developed world but occurs more frequently in the developing world.[1] Renal cortical necrosis commonly presents in the early childhood period and in the adult where the most common is the obstetric causes (50%-70%).[2,3,4] Non-obstetric causes (20%-30%) occurs due to various reasons, of which shock is most frequent and is having poor outcome.[4] The main predisposing factor is prolonged impairment of renal perfusion. Here we are presenting a case of young male who developed irreversible acute kidney injury, secondary to gastroenteritis and severe dehydration.

Case Report

A 17 year old boy was admitted with history of sudden onset of multiple episode of loose motion without associated fever, malena or bloody stools. There was no significant history of chronic illness or medication in past. This acute condition was complicated by sudden decrease in urine output just after three days of presentation, associated with renal dysfunction and microscopic haematuria for which the patient received multiple sessions of intermittent haemodialysis and proper antibiotics therapy. Despite best conservative management and renal replacement therapy, there was no any clinical improvement.

Lab investigations

Baseline investigations were suggestive of anemia, leukocytosis (predominantly neutrophils) but the peripheral smear did not show any abnormal cells, Serum lactate dehydrogenase was mildly raised (306 mg/dl). Liver function tests were normal throughout the course of illness. Autoimmune profiles including antinuclear antibody, anti-double stranded DNA antibody, anti-neutrophilic cytoplasmic antibody, anti-glomerular basement membrane antibody and rheumatoid factor were negative. Viral markers were negative, complement 3 & 4 and anti-streptolysin O tire were also within normal range. Serum procalcitonin level was raised. Urine examination suggestive of sub-nephrotic range proteinuria of 1.24 gm/ 24hr urine, persistent microscopic hematuria and leucocyturia without dysmorphic RBCs. 2D Echocardiography showed dilated LA and LV, global LV hypokinesia and LVEF 35% TO 45%.

Imaging study

Ultrasonography (USG) was suggestive of large globular kidneys (Rt kidney 12.1x 5.8 cm and Lt kidney 12x5.5 cm – Figure 1) with gross ascites. Non-contrast CT scan was suggestive of bilateral large

bulky heterogeneous kidneys with ill-defined hypodense areas without any obstructive lesions.

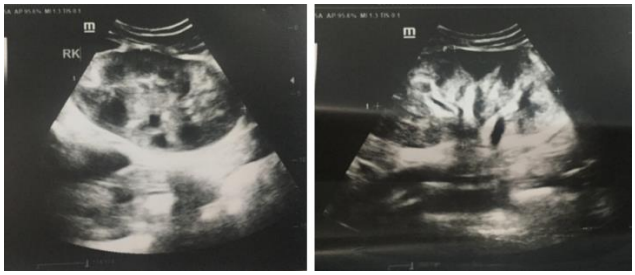

Fig. 1: USG showing bilateral large and bulky kidneys

Due to clinical instability, persistant uraemic symptoms and derranged renal profile, kidney biopsy was delayed. Later on, after receiving informed detailed consent, kidney biopsy was performed.

Histopathology

After receiving informed and detailed consent, kidney biopsy was performed. Light Microscopy (LM) suggested 50%-60% of glomeruli showed severe ischaemic changes with retracted tufts, mesangiolysis and segmental adhesions with confluent areas of deep cortical necrosis in 30% to 35% of sampled cortical areas (Figure 2 & 3). Direct immunofluorescence (DIF) was not significant, included non-viable cortical areas. Electron microscopy (EM) suggestive of partially necrosed cortical areas. viable areas reveal significant subendothelial rarefaction and accumulation of granular debris in subendothelial regions. No electron dense deposits seen and several tubules show electron lucent cytoplasmic vacuoles.

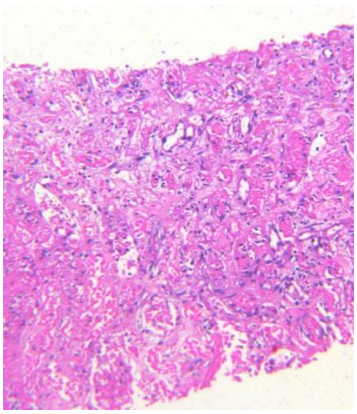

Fig. 2: LM (H&E stain X 63) - showing an area of confluent cortical necrosis

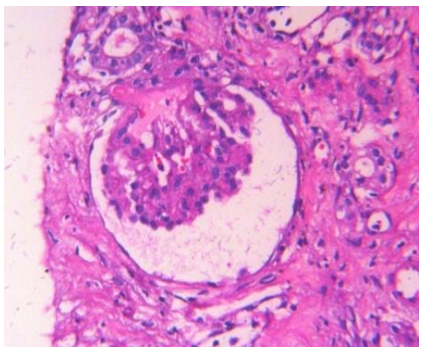

Fig. 3: LM(H&E X160) Glomerulus showing ischemic retraction of capillary tuft

Discussion

The predominant symptom of renal cortical necrosis is anuria or oliguria which is directly related to the fraction of the glomeruli affected.5,6 This patient presented with sudden onset of anuria, urine output of less than 50 ml/day throughout the course with persistant microscopic hematuria and nonselective subnephrotic protienuria. Lab parameters confirmed acute kidney injury,, leucocytosis and negative autoimmune profile with normal to enlarged kidney size. Despite regular haemodialysis and proper ultrafiltration, patient did not improve clinically and complicated with persistant volume overload. 2D ECHO was performed and it showed dilated LA/ LV, global LV hypokinesia and LVEF 35% -40%. Overall clinical and histopathological findings are suggestive of acute renal cortical necrosis. Irreversible damage to renal parenchyma leads to severe kidney injury and require renal replacement therapy[7,8] Correction of precipitating factor is the main concern to prevent acute kidney injury further progress to chronic kidney disease.

Conclusion

Renal cortical necrosis is an uncommon cause of acute kidney injury. Although obstetric causes are more common but nonobstetric causes like severe dehydration and persistant hypovolemia may also lead to severe acute renal cortical necrosis. Patients may develop severe renal dysfunction after several year of cortical necrosis but here, in our case, the disease was very catastrophic in nature. Patient did not respond to treatment and ultimately leads to permanent loss of

renal function. The only measure is to early recognition and prevention of precipitating factors.

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Declaration of Consent
Appropriate written consent were obtained before any procedure. All due efforts will be made to conceal the identity of patient but anonymity can not be guaranteed

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Conflict of Interest
None.

References

1. Grunfeld JP, Gaveval D, Bournerias F. Acute renal failure in pregnancy. *Kidney Int* 1980;18:179-91.
2. Matlin RA, Gay NF. Acute cortical necrosis: case report andreview of the literature. *Am J Med* 1974;56:110-8.
3. Kleinknecht D, Grunfeld JP, Cia Gomez P, Moreau JF, Garcia-Torres R. Diagnostic procedures and long termprogress in bilateral cortical necrosis. *Kidney Int* 1973;4:390.
4. Chugh KS, Singhal PC, Kher VK. Spectrum of acute cortical necrosis in Indian patients. *Am J Med Sci* 1983;286:10-20.
5. Levinsky NG, Alexander EA. Acute renal failure. In: Brenner BM, Rector FC, (eds) The kidney. 2nd edn, Philadelphia: WB Saunder Company, 1981; pp L51-NlBl.
6. 6.Rieselbach RE, Klahr S, Bricker NS. Diffuse bilateralcortical necrosis. A longitudinal study of the functionalcharacteristics of residual nephrons. *Am J f Med* 1967; 42:456-68.
7. Gelfand MC, Friedman EA. Prognosis of renal allotransplantsin patients with bilateral renal cortical necrosis. *Transplant* 1970;10:442-6.
8. American College of Surgeons/National Institute of Health. Transplant Registry, October 1976.

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