ORIGINAL RESEARCH PAPER
Volume-6 | Issue-11 | November-2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE OF ACUTE FOCAL RENAL CORTICAL NECROSIS DUE TO HUS IN ADULT PATIENT

Medicine

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KEYWORDS

1. Introduction:

Acute renal cortical necrosis is a rare cause of acute renal failure secondary to ischemic necrosis of the renal cortex. Majority of the patients become dialysis dependent and occasional patients may recover partial kidney function and are dialysis-independent. Acute cortical necrosis is usually a bilateral condition, rarely being unilateral (Blau et al.,1971). The lesions are usually caused by significant prolonged diminished renal arterial perfusion secondary to vascular spasm, micro-vascular injury, or intravascular coagulation. Renal cortical necrosis is usually extensive although local or localized forms occur.Most of the patients present as acute renal failure and suspicion of the condition arises following prolonged oliguria and/or anuria. The kidney biopsy is the gold standard for the diagnosis.

2. Case Report:

Mr. Mahesh Thakor, a 22 year old Indian patient was admitted on 18/9/2016 in ER of VSGH, Ahmedabad.Patient had history of fever since 9 days , decreased urine output since 8 days with abdominal pain and red coloured urine since last 5 days. Patient was given antibiotic (Cefexime) for 5 days and paracetamol for the same period by general practitioner from Dakor . Patient recoverd from fever but then he developed abdominal pain , reduced urine output with red coloured urine. And then admitted to VSGH.

He had a pulse rate of 70/min, respiratory rate of 14/min and his blood pressure was 116/70 mm Hg with saturation of 99% on room air , RBS – 134 mg/dl . Physical examination revealed soft per adbdomen , bilateral asymmetrical oedema of the lower limbs, with pallor.

Biological investigations at admission revealed: anaemia microcytic hypochromic with haemoglobin at 8.5 g/dl, thrombocytopenia 22900/mm3, leococytes 7280/mm3, creatinine 11.64 mg/dl, blood urea 255 mg/dl, serum calcium 8.0 mg/dl and phosphate 3.18 mg/dl. In blood electrolytes, sodium was 117 mmol/l, potassium 5.2 mmol/l. Transaminases were normal with AlkP 84 U/l, and ALT 43 U/l. blood sugar was 134 mg/dl. (**Table 1**).

Table 1. Showing laboratory values.

Laboratory parameter	Result	Normal value
Haemoglobin (g/dl)	8.5	12-16
Total leucocytes count (/mm3)	7280	4000-11000
Platelet count (/mm3)	22900	1.5-4.5 lakh
Blood urea (mg/dl)	255	15-45
Serum creatinine(mmol/l)	11.64	0.6-1.5
Serum sodium (mmol/l)	117	136-145
Serum potassium(mmol/l)	5.2	3.5-5.5
AlkP (U/l)	84	40-150
ALT (U/l)	43	0-55
Blood sugar(mg/dl)	134	70-110
Serum calcium(mg/dl)	8.0	8.4-10.2
Serum phosphorus(mg/dl)	3.18	2.30-4.70

An abdominal ultrasound showed normal sized kidneys with normal cortico-medullary differentiation.

Treatment consisted of: inj cefaxone+sulbactum 3gm/day for 10 days plus inj metronidazole 1.5gm/day in divided doses for 14 days alongwith inj NS(0.9%) ~2 l/day. Diuretics were avoided because complete anuria for first 4 days after admission. Because of complete anuria and worsening of acidosis patient was taken for hemodialysis on 2nd day and total 10 cycles of hemodialysis was done from which patient was on daily dialysis for a week and patient was transfused of 4 PCV and 6 PRC. Secondarily, evolution during hospitalization was favorable with a recovery of diuresis to 2 liters per day, a partial improvement of renal function (creatinine decreased from 11.64 mg/dl on 18/09/2016 at 4.07 mg/dl on 9/10/2016) and a normalization of the platelets count. Normocytic normochromic anemia persisted to 11.5 g/dl, however.

Given the persistence of impaired renal function, renal biopsy was taken which showed renal parenchyma with cortical necrosis of 2 glomeruli. The remainder one glomerulus show mild mesnagiolysis, normal capillary basement membrane and capillary Lumina. Focal area of necrosis of tubules, occasional foci of mononuclear inflammatory cells infiltration and interstitial edema are present. The medium sized blood vessels reveal unremarkable morphology with diagnosis of focal cortical necrosis.

Patient was discharged with serum creatinine of 2.42 mg/dL and around 2 liters of urine output and later on follow up, patient had normal RFT.

3.Discussion:

Our patient had presented with anuria , raised serum LDH and low platelet count , which raised suspicion of Hemolytic Uremic Syndrome (HUS). Renal ultrasonography was performed in our patient. It objectified normal size of kidneys with normal cortico-medullary differentiation. Renal biopsy was performed which revealed Focal Cortical Necrosis. After ruling out several causes of Renal cortical necrosis on the basis of history , clinical examination and relevant investigations, we considered HUS as the underlying cause of focal cortical necrosis in this patient.

Renal Cortial Necrosis is a rare clinicopathological AKI form. It consists of a bilateral ischemic necrosis, symmetrical, “patchy” diffuse to renal cortex, sparing the renal medulla and a thin strip of subscapular cortex . Confirmation was made by renal biopsy, although this is not necessary for retaining the diagnosis of RCN. Other paraclinical investigations such as renal Doppler, “microbubble” ultrasound or ultrasound contrast, abdomen and pelvis scan or renal MAG3 scintigraphy [14] may be sufficient.

This patient improved with supportive therapy and 6 cycles of dialysis , not requiring maintainance dialysis. The prognosis of RCN is often pejorative, requiring the use of chronic haemodialysis, apart from cases where segmental renal cortical necrosis and delayed partial recovery can be observed.

In untreated patients, the mortality rate from renal cortical necrosis exceeds 50%. Early initiation of dialysis significantly diminishes this rate.The most important prognostic factors are the extent of necrosis, duration of oliguria, and severity of associated conditions.

4.References:

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