Original articles

Spectrum of renal cortical necrosis in acute renal failure in Eastern India

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Summary

Renal cortical necrosis is an uncommon cause of acute renal failure. We report 23 cases of biopsy-proven renal cortical necrosis which constituted 6.3% (23/363) of all cases of acute renal failure studied over a period of seven years (1985–92). The patients were divided into two groups: obstetric and non-obstetric. Obstetric complications were responsible for renal cortical necrosis in 15 (65.2%) patients while non-obstetric conditions accounted for the remaining eight (34.8%) cases. The overall incidence of cortical necrosis in obstetric acute renal failure was 15/63 (23.8%) patients, the incidence being nearly equal in early (20.5%) and late (29%) pregnancy. Post-abortion renal failure was the sole cause of cortical necrosis in early pregnancy in the obstetric group. Haemolytic uraemic syndrome (three patients) and septicæmia (two patients) were the main cause of necrosis in the non-obstetric group. The cortical necrosis was diffuse and patchy in 17 and six patients, respectively. The disease had a fatal prognosis in 20 (87%) patients; mortality was due to uraemic complications and infections in the majority of patients. The high frequency of post-abortion renal cortical necrosis in our patients is similar to the experience of other Indian workers.

Keywords: septic abortion, acute renal failure, cortical necrosis, India

Introduction

Acute bilateral renal cortical necrosis is a rare but potentially fatal variety of renal lesion. There is global necrosis of all of the elements in the involved area of the cortex, including blood vessels, glomeruli, and tubules. Irreversible loss of kidney function is the rule in complete cortical necrosis but is variable in the incomplete type where less than 50% of nephrons are necrotic. Renal cortical necrosis is an uncommon entity that accounts for only 2% of all causes of acute renal failure.¹ Obstetric complications are the commonest (50–70%) cause of renal cortical necrosis^{2,3}; non-obstetric causes account for 20–30% of all cases of cortical necrosis and in these circumstances the incidence is higher in men than in women.⁴ The

non-obstetric conditions leading to acute cortical necrosis are: extensive burns, pancreatitis, septicæmia, snake bite, and diabetic keto-acidosis.^{5,6} Abruptio placentæ, septic abortion, eclamptic toxæmia, post-partum hæmorrhage and puerperal sepsis are the pregnancy-related situations responsible for renal cortical necrosis.^{1,7} The purpose of this paper is to study the causes, clinical course, and outcome of renal cortical necrosis in patients with acute renal failure.

Methods

Over a period of seven years (1985–92), 23 patients had biopsy-proven renal cortical necrosis out of a total of 363 cases of acute renal failure studied during this period. A complete history and physical examination findings with special emphasis on duration of oliguria/anuria, hypotensive episodes, sepsis/septic shock and severity of renal failure were noted in each case. Laboratory investigations included estimation of urea, creatinine, total and differential leucocyte count, hæmoglobin, serum uric acid, calcium, phosphorus, and alkaline phosphatase. Haematological and coagulation studies were conducted to rule out hæmolysis and disseminated intravascular coagulation as and when required. Renal tissues were obtained by a percutaneous approach (necropsy in 15, biopsy in eight) in all patients and studied using light microscopy. Renal biopsy was done in eight patients with prolonged renal failure (oligoanuria lasting more than five weeks). The patients showing cortical necrosis on histology were selected for the present study. Based on the following criteria, patients were divided into two groups: *Complete cortical necrosis*: essentially irreversible renal failure. Confluent global cortical destruction extending into the columns of Bertin. Thin rim of subcapsular and juxta-medullary tissue being preserved. *Patchy cortical necrosis*: contiguous area of cortical necrosis up to one-third to one-half of entire cortical tissue. This form has potential for partial recovery of renal function.

Results

Of 363 patients with acute renal failure, 23 (6.3%) had renal cortical necrosis. The patients fell into two groups: 15 obstetric (65.2%), and eight non-obstetric (34.8%). All patients were initially treated at peripheral centres (primary

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Accepted 30 November 1994

health centres or private practitioners) and were referred to us only after the development of acute renal failure with a duration of oligoanuria of 4–10 days. These patients had severe acute renal failure on admission with neuropsychiatric manifestations (11), metabolic acidosis (four), bleeding diathesis (two), and fluid overload (five). The severity of renal failure is shown in table 1. All patients were treated with dialysis (peritoneal/haemodialysis) in addition to supportive treatment using standard criteria until they improved or died. The obstetric complications leading to cortical necrosis are shown in table 2. The cortical necrosis was observed in 15/63 (23.8%) patients with obstetric acute renal failure. The incidence of cortical necrosis in early and late pregnancy was nearly equal, 20.5% and 29%, respectively. In the obstetric group, three patients (20%) had patchy and 12 (80%) had diffuse renal cortical necrosis. There were eight cases (seven males; one female) in the non-obstetric group. The clinical features of the non-obstetric conditions leading to cortical necrosis are given in table 3. One patient survived with partial recovery of renal function in the obstetric group and two in the non-obstetric group.

The overall mortality was 20 (87%). Death in 11 patients was attributed directly to uraemia, as the patients could not afford dialysis due to financial constraints. Uraemic complications leading to death were: profound azotemia (six), pulmonary oedema (three) and hyperkalaemia

in (two) patients. Septicaemia was another major cause of mortality in eight (34.8%) patients and one death was due to gastrointestinal haemorrhage. The majority of deaths occurred in the first two weeks (15/23) and necropsy specimens of renal tissue were obtained in all such cases for histological studies.

Discussion

Renal cortical necrosis is a rare entity that accounts for only 2% of all cases of acute renal failure.^{1,3} Our study revealed 23 (6.3%) patients had renal cortical necrosis among 363 patients with acute renal failure. The higher incidence of cortical necrosis in our patients with acute renal failure was possibly related to more frequent occurrence of cortical necrosis in post-abortion renal failure. Obstetric complications are responsible for 50–70% of cases of renal cortical necrosis in most reported series.^{1,3,7} We observed 65.2% of cases of obstetric origin. Abruptio placentae is the most common obstetric situation associated with cortical necrosis.^{2,4} The peak incidence is in the last trimester of pregnancy and affects older, multiparous women. It is not clear why most victims of abruptio placentae who develop acute renal failure have acute tubular necrosis (80%), whereas only a minority suffer from cortical necrosis.

Toxaemia of pregnancy, uterine haemorrhage, and puerperal sepsis are other conditions associated with cortical necrosis in late pregnancy.^{7,8} Renal cortical necrosis is rare following septic abortion.⁹ The incidence of cortical necrosis in early pregnancy (post-abortum group) was 20.5% compared with 29.2% in late pregnancy. The high incidence of renal cortical necrosis in early pregnancy is the interesting feature of our study. Similar observations have been reported in an earlier report from this country.¹⁰ Thus we observed two peak incidences of obstetric cortical necrosis; one following septic abortion and the other in late pregnancy, in contrast to the findings in developed countries. Cortical necrosis unrelated to pregnancy accounts for 20–30% of all cases, and in a non-obstetric setting a higher incidence is observed in males than in females.⁴ There was only one woman in eight cases of non-obstetric cortical necrosis in the present study. In most cases, a combination of shock (either hypovolemic or septic) and disseminated intravascular coagulopathy is usually present. Four patients out of eight in our study had hypotension and shock in association with burns, sepsis, and massive gastro-intestinal haemorrhage. Patchy cortical necrosis was reported in one patient following diarrhoea. This patient had a partial recovery of renal function and is surviving without the need for dialysis. The anuria of cortical necrosis (0 to 50 ml/day) is the striking feature in contrast to the 400 to 500 ml/day output in acute tubular necrosis.¹¹ Nineteen of 23 patients had absolute anuria and in four cases, 24-hour urinary output was less than 100 ml. The duration of anuria varied from 4–14 weeks. Evidence of

Table 1 Severity of renal failure (n = 23)

Data	Range	Mean
Day of referral (days)	4–10	5.3
Peak pre-dialysis urea (mg %)	(115–346.5)	158.2
Peak pre-dialysis creatinine (mg %)	9–23.6	16.8
Peak serum potassium (mEq/l)	4.2–7.8	4.6

Table 2 Obstetric causes of renal cortical necrosis (n = 15)

Obstetric group	n	%
Post-abortum	8	34.8
Post-partum haemorrhage	3	13.0
Intra-uterine death	2	8.7
Puerperal sepsis	2	8.7

Table 3 Non-obstetric renal cortical necrosis (n = 8)

Conditions	Age	Sex	Type of necrosis	Results
HUS	18 m	M	Diffuse	Died
HUS	8 yr	M	Diffuse	Died
HUS	6 m	M	Patchy	Survived
80% burns, shock	22 yr	F	Diffuse	Died
Pneumonia sepsis, shock	48 yr	M	Patchy	Died
Massive GI bleed, hypotension	36 yr	M	Patchy	Died
Septic shock	22 yr	M	Diffuse	Died
Post-diarrhoeal	18 yr	M	Patchy	Survived

HUS=haemolytic uraemic syndrome; GI=gastrointestinal

disseminated intravascular coagulopathy in the form of thrombocytopenia, hypofibrinogenemia, and prolonged prothrombin time was seen in seven and three patients in early and late pregnancy, respectively. The clinical course and outcome of patients with cortical necrosis can be classified into five broad groups (see box). Twenty (87%) patients died during the acute phase in our study. The various causes of mortality were severe uraemia (six), pulmonary oedema (three), hyperkalaemia (two), gastrointestinal haemorrhage (one) and septicaemia (eight) patients. Thus, the majority of deaths which occurred in the acute phase were due to septicaemia and uraemia in patients who could not afford dialysis. However, the prognosis and survival of patients with cortical necrosis has improved markedly in developed countries due to the availability of renal replacement therapy.^{12,13} Three patients had a partial recovery of renal function and are surviving without dialysis support at six months follow up. They had prolonged oliguria (more than 35 days) and biopsy revealed patchy cortical necrosis. In certain patients, there may be a slow rise in creatinine clearance and a gradual gain in renal function over one to two years, so that the glomerular filtration rate may reach a final plateau level of approximately 20 to 25 ml/min.^{14,15} It is assumed that juxtamedullary glomeruli (which comprise 15 to 20% of the total) escape destruction, even in complete cortical necrosis and that early functional return is due to recovery of these nephron segments. A significant number of patients may develop deterioration in renal function several years (1 to 10 years) after cortical necrosis.³ Factors causing this late functional downturn are not clear but include pyelonephritis, hypertension, and shrinkage of the kidneys due to progressive fibrosis and/or calcification.^{16,17}

Acute bilateral cortical necrosis is an uncommon disease at the extreme end of the spectrum. Its incidence and severity has declined in obstetric practice in developed nations. In contrast to western countries renal cortical necrosis is still high in obstetric acute renal

Cortical necrosis: clinical course

- death in uraemia during the acute phase
- survival without dialysis
- late return to dialysis/transplantation
- survival only with chronic maintenance dialysis/transplantation
- late resumption of sufficient renal function to become dialysis independent

Summary/learning points

- acute renal failure in pregnancy is still high in India
- obstetric cortical necrosis has two peaks; in early and late pregnancy
- the incidence of cortical necrosis is high in post-abortion acute renal failure

Causes and clinical feature of renal cortical necrosis

- septic abortion
- haemolytic uraemic syndrome
- septicaemia with shock
- absolute anuria
- severe uraemic complications
- high mortality

failure in Indian patients. The high incidence of cortical necrosis in early pregnancy is a striking feature of this study as its occurrence is infrequent in early pregnancy in western countries. Thus, obstetric cortical necrosis had two peaks in our patients, one in post-abortion renal failure and the other in late pregnancy. The disease carries a high mortality, chiefly due to poverty and non-availability of chronic dialysis in this part of the country. The causes, clinical feature and learning points related to renal cortical necrosis are given in the boxes.

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