RENAL CORTICAL NECROSIS: THREE CASE REPORTS AND A REVIEW OF THE LITERATURE

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İ.Ç.Özener, MD*/R.A.Lawrence, MD**/E. Akoğlu,M.D.***

* *Professor, Department of Internal Medicine, Faculty of Medicine, Marmara University, Istanbul, Türkiye.*
** *Assistant Professor, Department of Internal Medicine, Faculty of medicine, Marmara University, Istanbul, Türkiye.*
*** *Specialist, Department of Internal Medicine, Faculty of Medicine, Marmara University, Istanbul, Türkiye.*

SUMMARY

Acute bilateral renal cortical necrosis is a rare condition most commonly occuring as a complication of pregnancy. It is characterised by oligoanuria and severe acute renal failure requiring dialysis if the patient is not to die. This paper describes three cases of renal cortical necrosis and briefly reviews the literature.

Key Words : Renal cortical necrosis, acute renal failure

INTRODUCTION

Acute bilateral renal cortical necrosis was first described in 1886 by Juhel-Renoy following the autopsy of a sixteen year old girl dying of scarlet fever following a period of oliguria (1). Ten years later Bradford and Lawrence reported the association of renal cortical necrosis (RCN) and pregnancy (2). Up until 1971 when the last major review was written over 400 cases had been described (3).

Despite progress in the diagnosis of renal disease in recent years the incidence of RCN has decreased (4), and reflects improved quality of patient care.

Here we describe 3 cases of RCN presenting over a nine year period between 1982 and 1991. In the same period 142 cases of acute tubular necrosis were seen.

CASE REPORTS

CASE I. A thirty three year old previously well female patient presented to maternity hospital in the ninth month of her sixth pregnancy complaining of inguinal pain, fever, and rigors, and then delivered a stillborn child. The following day the patient developed nausea, vomiting, and diarrhoea, with concommitant jaundice. On the third day anuria occured, and the blood urea nitrogen (BUN) was estimated at 180 mg/dl. At this point the patient was referred to our hospital.

On admission the patient was pale, jaundiced and confused. Her body temperature was 37 C, pulse rate 100/min., and her blood pressure 130/90 mm Hg. Chest examination revealed fine bilateral crepitations and scattered wheezing. The liver was firm and palpable 2 cm below the costal margin. A flapping tremor of the hands was present. Other physical findings were unremarkable.

Laboratory testing revealed the following; Hb 9 gm/dL, Hct 29%, WBC 12.200/mm, BUN 135 mg/dL, serum creatinine 8 mg/dL, SGOT 400 IU/L, SGPT 120 IU/L, total bilirubin 28.5 mg/dL, unconjugated bilirubin 13.7 mg/dL, serum sodium 130 mEq/L, serum potassium 5.1 mEq/L, and serum chloride 98 mEq/L.

A diagnosis of acute tubular necrosis and sepsis was made and peritoneal dialysis was commenced. The patient died on the fifth day following admission. A post mortem biopsy of liver and kidney was performed and revealed the presence of diffuse renal cortical necrosis and non specific inflammatory changes in the liver.

CASE 2. A 30 year old previously well female patient in the sixth month of her seventh pregnancy presented to a local maternity hospital complaining of dysuria, nocturia, and oedema of the hands, feet, and periorbital region. Her blood pressure was reported to be high but only salt restriction was implemented as therapy. At the end of the ninth month of her therapy she was readmitted in shock and complaining of abdominal pain when a diagnosis of placeta abruptio was made. She was taken for surgery where a subtotal hysterectomy was performed. Two days into the postoperative period anuria occured and the patient was referred to our department.

On admission the patient was pale and shocked with a blood pressure of 70/40 mm Hg. She was tachycardic with a pulse rate of 108 beats/min. but apyrexial with a body temperature of 37.5 C.There was abdominal and costovertebral angle tenderness but otherwise physical examination was unremarkable.

Laboratory testing revealed the following; Hb 8 gm/dL, Hct 25%, WCC 8200/mm, BUN 72 mg/dL, creatinine 7.9 mg/dL, serum sodium 136 mEq/L, serum potassium 5.3 mEq/L, and serum chloride 102 mEq/L. Microscopy of the urine sediment revealed 9-10 erythrocytes and 3-4 leucocytes per high power field. The urinary sodium concentration was 100 mEq/L. Urine culture demonstrated the presence of Pseudomonas sp. and antibiotic therapy with Tobramycin and Clindamycin was prescribed.

The patient's urine output over the previous few days was estimated to have been 20-100 ml, and her acute renal failure was treated with peritoneal dialysis. The patient remained oliguric for one month at which stage her urine output started to rise. Two weeks later the patient was discharged with good urine output and a BUN of 38 mg/dL and a serum creatinine of 3.2 mg/dL. A plain abdominal film obtained a month after discharge was pathognomic of RCN, bilateral cortical calcification being present in both kidneys. The patient was treated conservatively in the renal outpatient department for one year before being lost to follow up.

CASE 3. This patient was a 46 year old female in the seventh month of her eighth pregnancy, and again the previous medical history was unremarkable. The presenting complaint to the maternity hospital was periorbital and pedal oedema. One month later the patient miscarried and delivered a stillborn child. The patient became oliguric and three days later was referred to our unit.

On admission the patient was apyrexial with a body temperature of 37.2 C. The patient was fluid overloaded with a blood pressure of 210/120 mm, a pulse rate of 130 beats/min., distended neck veins, bilateral basal crepitations of the lungs, a liver palpable 4 cm below the costal margin, and bilateral pitting oedema of the ankles. The patient appeared pale and anaemic and a 2/6 functional systolic ejection murmur was heard in the pulmonary area of the heart. Both costovertebral areas were tender.

Laboratory tests were as follows; Hb 6.6 gm/dL, WCC 6800/mm, BUN 63 mg/dL, serum creatinine 9.6 mg/dL, serum sodium 138 mEq/L, serum potassium 4.5 mEq/L, and serum chloride 94 mEq/L. The urinary sediment contained many leucocytes and renal tubular cells.

A hysterectomy was performed, and haemodialysis commenced. The oliguric phase had lasted 45 days when the patient started to pass urine. At discharge the daily urine volume was 1500 ml and the creatinine clearance 15 ml/ml when dialysis was discontinued .Three months later a plain abdominal. film showed bilateral renal calcification. At one year follow up the creatinine clearance had risen to 25 ml/min.

DISCUSSION

Cortical necrosis is an uncommon form of acute renal failure (ARF) as evidenced by the paucicity of reports in the literature in recent years. In 1973 Kleinknecht et. al. reported RCN to occur in approximately 2% of cases with acute renal failure (5). RCN has been reported to occur in both sexes and at all ages with cases reported in infancy and childhood including onset in the antepartum period (3). It is however much commoner in pregnancy, fifty percent of cases reported in the earlier literature relate to gestation and in their series of 38 patients Kleinknecht et. al. reported the incidence of obstetric cases to be 70% (5). The incidence of RCN during pregnancy, like that of all forms of ARF, is in the West at least declining. Thus, in one study carried out in Dublin, the incidence of RCN was one in 10.000 deliveries between 1961 and 1970 but had dropped to one in 80.000 between 1971 and 1980 (4).

RCN occurs most commonly after placental abruption and has been reported to be present in approximately half of all obstetric cases (3), and is less common in cases of intrauterine death or as a consequence of eclampsia (6). Amongst patients with placental abruption the frequency of RCN is related to age being greater in patients over 30 years of age (3). Within this group frequency is not related to parity but occurs most commonly between the twenty third and thirty first weeks of pregnancy (3)

The pathophysiology of RCN remains unclear but in most cases an underlying pathology or physiological stress (pregnancy) is present.

Conditions reported by Woods and Williams (3) to be associated with the onset of RCN include;

I) Pregnancy, often but not always associated with preeclampsia, with;
a) Premature placental separation and concealed haemmorhage
b) Septic abortion and other infections
c) Nonseptic abortion
d) Placenta praevia
e) Post partum haemmorhage
f) External puerpral haemmorhage and shock
g) Renal vein thrombosis
h) Diabetes
i) Fatty liver
j) No abnormality other than eclampsia

II) Cases in infancy and childhood associated with;
a) Vomiting, diarrhoea and usually fever
b) Infections: Pharyngitis, tonsilitis, scarlet fever, tuberculosis, peritonitis, staphylococcal septicaemia, and pneumonia
c) Transfusion reactions
d) Shock following traumatic delivery
e) Maternal antepartum haemmorhage
f) Phosphorous poisoning

III) Cases occuring in adults but not associated with pregnancy;
a) Infections: tonsilitis, pneumonia, tuberculosis and peritonitis
b) Vomiting and/or diarrhoea
c) Shock
d) Poisoning: alcohol, ethylene glycol, and cadmium
e) Snake bite
f) Haemolytic anaemia associated with thrombocytopaenia
g) Heart failure
h) Burns

Clinically patients with acute cortical necrosis may be alert and feel well at the time of diagnosis. Abnormal

physical findings may be minimal although there may be some tenderness of the costovertebral angles. Urine output is usually very low and may be zero in the first few days often increasing to 300 to 500 ml but rarely more. Urinanalysis when available shows marked albuminuria and gross or microscopic haematuria. White cells may be present but casts are rare. These patients without dialysis develop uraemia from which they ultimately die. Prior to the advent of haemodialysis in 1955 RCN was invariably fatal (3).

The onset of anuria may occur without any clinical evidence of shock or hypotension although this is present in most patients with placental abruption. In many patients with RCN secondary to infection there has been no demonstration of a reduction or redistribution in blood volume, and the presence of shock or hypotension is not a constant finding (3). The pathogenesis of RCN is uncertain although most authors agree that the mechanism is ishaemic. The factors responsible for this ischaemia are unclear but the underlying response is considered to be a sustained vasospasm of the interlobar and interlobular arteries followed by thrombosis and coagulation necrosis. It has been suggested that transitory vasospasm lasting only a few hours may be sufficient to induce RCN (7). Vasospasm does not always appear to be uniform throughout the kidney (8,9) resulting in patchy necrosis and sparing enough cortex in some cases to sustain life (10).

Renal size increases for two to three weeks after the onset of anuria but if the patient survives size diminishes (11). The capsule of the kidney is not thickened, and almost invariably I to 2 mm of cortical tissue underlying the capsule remains viable. The medulla although congested with blood appears normal (12). Necrotic lesions of the cortex may range in severity from small I mm foci to gross necrosis of the entire cortex (12).

Microscopically areas of complete necrosis are characterised by the death of all cell types without distortion of basic structural details (13). Glomerular capillaries are distended with dehaemoglobinised cells, tubular cells are pyknotic and strip away from the basement membrane. Afferent arterioles contain fibrin and platelet thrombi. This thrombus may extend backwards into the intralobular and arcuate arteries (3). Calcification within the necrotic renal parenchyma has been identified pathologically as early as the fifth day (14) and in patients surviving past three weeks radiological evidence of calcification is usually present (3) (see Fig. I). It may outline the borders of the necrotic tissue to give a double or "tramline" appearance (10).

The three cases of RCN presented here are all pregnancy related being secondary to septic abortion, placental abruption and preeclampsia with spontaneous abortion respectively. The exact nature of the event provoking the abortion in the third patient was never clear.

RCN is a frequently fatal disease which is frequently diagnosed after irreversible renal failure has occured and the best from of treatment is prophylaxis. This involves better standards of patient care particularly obstetric care.

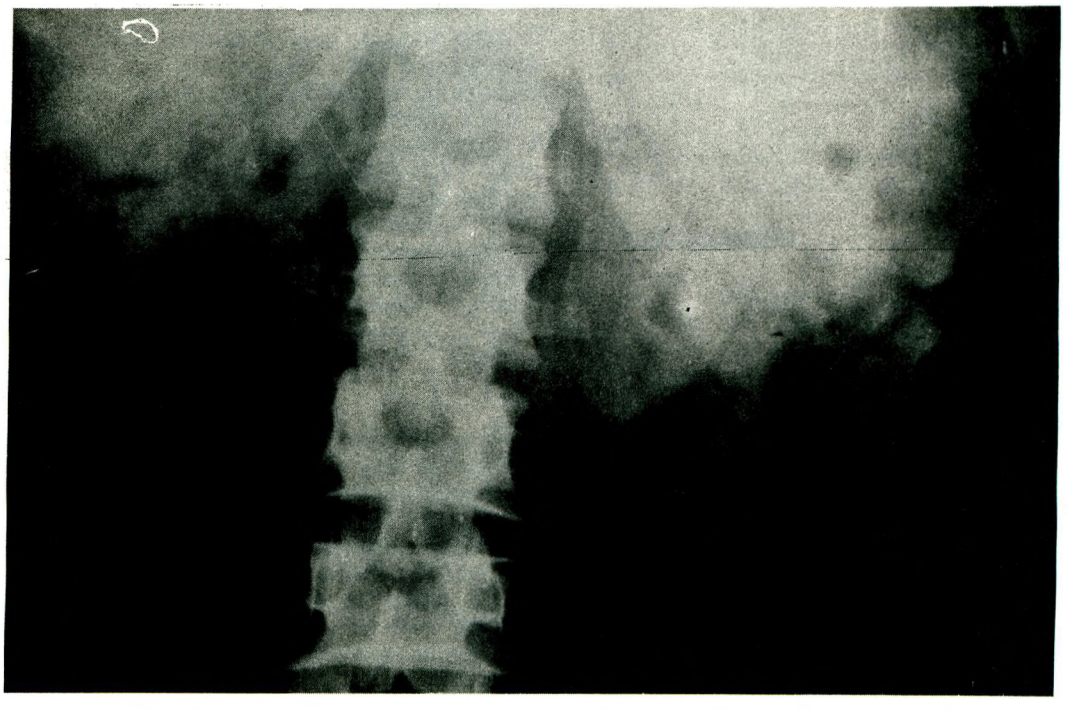

Fig.1. Plain abdominal film of patient 2 showing bilateral cortical calcification and double outlining.

REFERENCES

1. Juhel-Renoy E. De Lànurie précoce scarlatineuse. Arch Gen Med VII serie 1886; 17:385.
2. Bradford JR, Lawrence TWP. Endarteritis of the renal arteries, causing necrosis of the entire cortex of both kidneys. J Path Bact 1898; 5:195.
3. Woods JW, Fraklin Williams T Diseases of the kidney. In: Strauss MB, Welt LG, eds. Hypertension due to renal vascular disease, renal infarction, renal cortical necrosis. Boston: Little Brown, 1971:769.
4. Madias NE, Donohoe JF, Harrington JT. Post-ischemic acute renal failure. In: Brenner BM, Lazarus JM, eds. Actue renal failure. 2 nd ed. New York: Churchill-Livingstone, 1988: 597.
5. Kleinknecht D, Grunfeld JP, Gomez PC, Moreau JF Gracia-Torres R. Diagnostic procedures and long-term prognosis in bilateral renal cortical necrosis. Kid Int 1973; 4:390
6. Lidheimer MD. Katz AI. The kidney and hypertension in pregnancy. In: Brenner BM, Rector FC, eds. The Kidney. 4th ed. Philadelphia: WB Saunders, 1991:1551.
7. Davis JC. Case records of the Massachusetts General Hospital. New Eng J Med 1958; 258:1219.
8. Boucet NG, Guild WR, Merrill JP. Bilateral renocortical necrosis with recovery: Report of a case. N Eng J Med 1957; 257:416.
9. Gormsen H, Iverson P, Raaschou F. Kidney biopsy in acute anuria: with a case of actue, bilateral cortical necrosis. Am J Med 1944; 19:209
10. Lloyd-Thomas HG, Balme RF, Key JJ. Trim-line calcification in renal cortical necrosis. Br Med J 1962; I:909
11. Moell H. Gross bilateral renal cortical necrosis during long periods of oliguria-onuria: Roentgenologic observations in two cases. Acta Radiol 1957; 48:355.
12. Sheehan HL, Moore HC. Renal Cortical Necrosis and the kidney of concealed accidental hemorrhage. Springfield III. Thomas, 1953:135.
13. Lang EK. Arteriographic diagnosis of renal infarctions. Radiology 1967; 88:1110.
14. Whelan JG Jr, Ling JT, Davis LA. Antemortem roentgen manifestations of bilateral renal cortical necrosis. Radiology 1967;89:682.