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REVIEW ARTICLE

ACUTE RENAL CORTICAL NECROSIS

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ABSTRACT

Acute renal cortical necrosis is a rare cause of acute renal failure secondary to ischemic necrosis of the renal cortex. It accounts for only 2% of all causes of acute renal failure in developed countries (Grünfeld *et al.,* 1981), but occurs more frequently in developing world (Chugh *et al.,* 1976; Chugh *et al.,* 1983; Hassan *et al.,* 2009; Parkash *et al.,* 1995). The obstetric complications are the commonest (50 – 70%) cause of renal cortical necrosis (Hassan *et al.,* 2009), non-obstetric causes account for 20-30% of all cases of cortical necrosis. The classic description of this condition by Sheehan & Moore (1952) still holds true to this day. They described the lesion at different stages in evolution as seen in autopsy material. The pathogenesis of cortical necrosis is far from clear, and probably many factors are involved. Acute renal failure as a result of acute cortical necrosis cannot be distinguished readily from other forms of acute renal failure such as acute tubular necrosis and renal biopsy is the only sure way of making the diagnosis during life (Lauler & Schreiner, 1958). Partial recovery of renal function has been reported and it is likely that recovery is governed by the extent of the lesion (Walls *et al.,* 1968).

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INTRODUCTION

Acute renal cortical necrosis is a rare cause of acute renal failure secondary to ischemic necrosis of the renal cortex. It accounts for only 2% of all causes of acute renal failure in developed countries (Grünfeld *et al.,* 1981), but occurs more frequently in developing world (Chugh *et al.,* 1976; Chugh *et al.,* 1983; Hassan *et al.,* 2009; Parkash *et al.,* 1995). The obstetric complications are the commonest (50 – 70%) cause of renal cortical necrosis (Hassan *et al.,* 2009), 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 (Duff & More, 1941). 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.

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Etiology

Pregnancy related

- Abruptio placentae
- Severe pre-eclampsia / eclampsia
- Criminal / Septic Abortion (Gram negative septicemia)
- Hyperemesis gravidarum
- Prolonged intrauterine death

Infections

Children

- Diarrhea, vomiting (Dehydration)
- Peritonitis
- Septicemia
- Congenital heart disease

- Fetal maternal transfusion
- Dehydration.
- Perinatal asphyxia
- Placental hemorrhage
- Hemolytic uremic syndrome (HUS).

Adults & adolescents

- Scarlet fever
- Streptococcal Infections
- Peritonitis
- Cholera

Hemodynamic causes

Acute tubular necrosis progressing to acute cortical necrosis with shock and crush injury.

Trauma

Head injuries

- Burns
- Gastrointestinal hemorrhage
- Thrombotic thrombocytopenic purpura
- Pancreatitis
- Dissecting aneurysm

Snake bite: Due to direct toxic effect or shock, hemorrhage, haemoptysis

Drugs: Nonsteroidal anti-inflammatory drugs and contrast media.

Hyper acute kidney transplant rejection

Poisonous plants

Fava Beans

- Exposure to Sap of Marking-nut tree
- Almond Extract (? Cyanide)

Glycol poisoning

Dioxane

- Di ethylene Glycol (anti freeze)

Metallic & other poisoning

Arsenic

- Cadmium
- Lithium Carmine
- Pyrazolene
- Camphor
- Phosphorus

Idiopathic

In a small number of cases, no cause is apparent even after extensive search. These cases are
labeled as idiopathic in origin.

Pathology

The classic description of this condition by Sheehan & Moore (1952) still holds true to this day. They described the lesion at different stages in evolution as seen in autopsy material. They divided the fully developed form into various types, depending on the extent of the lesion.

Focal form

In this small scattered foci of necrosis are seen that vary from lesion of individual glomeruli to areas of cortical necrosis 0.5 mm in diameter. On gross examination kidneys usually are slightly enlarged and have punctuate red areas on cut section and on the sub capsular surface. Histologically, only few glomeruli in any one focus are affected, showing necrosis often with thrombosis at the vascular pole (Figure 1). Proximal convoluted tubules are always necrotic, and the distal tubules are affected similarly in the centre of larger lesions. In the remainder of the cortex, many proximal convoluted tubules appear necrotic, but glomeruli and distal convoluted tubules show no changes of consequence. There may be an overlap between acute tubular necrosis and cortical necrosis (Sheehan & Moore, 1952).

Minor form

The changes are similar to those described previously on gross description, except that lesions upto 3 mm in diameter are found. Grossly, the affected foci have white centers with a red congested rim. Histologically, in the affected foci there is necrosis of all elements including afferent arteriole, and interlobular artery. These and the glomeruli often contain thrombus material. Polymorphonuclear leucocytes are found sometime in portions of the necrotic lesion analogous to the peripheral dead zone of small infarct. Extensive proximal tubular necrosis is found in the remainder of the cortex.

Patchy form

Numerous larger polar areas of necrosis are found, sometimes occupying most of the width of the cortex, with a zone of congestion and hemorrhage around the periphery. The congestion is particularly pronounced in the inner cortex. The patches of cortical necrosis occupy about one third to two thirds of the cortex, but the columns of Bertini are usually spared. The kidneys are moderately enlarged. Histologically the foci of cortical necrosis are large enough to show a central dead zone. All the structures within the necrotic areas usually are necrotic although occasionally collecting ducts appear undamaged. The arteries and arterioles are necrotic and dilated, and contain thrombus material in cases seen more than 2 days after onset.

Gross renal cortical necrosis

The cortex is almost entirely necrotic with the exception of thin surviving areas immediately under the capsule & at the cortico-medullary junctions. The kidneys are usually enlarged when the condition is well developed. Almost the entire cortex is yellowish white except from the spared zones in the sub capsular and juxtamedullay cortex. If seen earlier, the affected cortex is somewhat hemorrhagic or congested with whitish yellow streaks. The columns of Bertini are necrotic. Histologically, changes similar to those seen in the patchy

form are seen, but with arteries showing necrosis and thrombosis over a greater length than in the patchy form (Figure 2).

Confluent focal cortical necrosis

This condition is very common in those not associated with abruptio placentae (Sheehan & Moore, 1952). In this type there are widespread lesions of glomeruli and tubules, but there is no involvement of arteries. The lesions vary greatly from nephron to nephron both in severity and in apparent age. The glomeruli either appear normal or show various changes such as congestion, thrombosis of capillaries or of the vascular pole, or frank necrosis. Manyproximal convoluted tubules appear normal but others are necrotic. Distal convoluted tubules appear normal. On gross inspection in the early stages the kidney has a red congested cortex with punctuate hemorrhages or pale mottling, but no white infracted areas.

Calcification in cortical necrosis

In some cases, in which partial recovery occurs & patient survives for several weeks/months calcification of necrotic cortex may occur which can be seen on radiology (Alwall *et al.,* 1958; Effersoe *et al.,* 1962; Oram *et al.,* 1964; Phillips, 1962).

Necrosis in other organs

Other organs may show necrosis in cases of renal cortical necrosis e.g., anterior lobe of pituitary, adrenals, spleen, lungs, gastro intestinal tract, liver, pancreas etc (Sheldon & Hertig, 1942).

Pathogenesis

The pathogenesis of cortical necrosis is far from clear, and probably many factors are involved.

Vasospasm

Following abruptio placentae, there is an initial vasospasm that reduces blood flow for periods varying from several minutes to six hours. The spasm then abates and recirculation of blood occurs. If the spasm is of short duration and good flow is re-established, acute tubular necrosis occurs. However in patients who develop cortical necrosis, a new spasm isthought to occur, this time more proximally in the vascular tree & lasting for upto 30 hours, causing necrosis of the arteries beyond the obstruction. Then thrombosis occurs, with permanent blockage to the circulation (Matlin & Gary, 1974; Schrieiner, 1979).It has long been suggested that vasculature in pregnancy is more prone than usual to vasoconstriction which may partly account for the greater frequency of cortical necrosis in this state.

Generalized Schwartzman reaction

Similarity has been shown between cortical necrosis & the generalized Schwartzman reaction in rabbits. In this reaction, two small doses of bacterial endotoxin given 24 hoursapart, cause microscopic thrombosis that involves the glomerular capillaries and leads to development of renal cortical necrosis (Moss *et al.,* 1977; Sporn, 1978). This mechanism maybe active during septic abortions.The sequence of events starts with sudden widespread dilation of glomerular capillaries, follows by an escape of plasma by filtration. Increased viscosity of the blood follows, with stasis and formation of thrombi, which extend backward to arteries of increasing size, which become necrotic.

Vascular thrombosis

The importance of fibrin and fibrinogen deposition in the glomeruli and small vessels has been demonstrated. This may result from mechanical obstruction with blood flow through the glomeruli. However, there is disagreement about the chain of events; whether coagulation or vasomotor dependence phenomena occur earlier. Endotoxaemia and/or bacterial sepsis is by far the most common factor responsible for intravascular coagulation. Immunologic mechanisms also may play a role in the pathogenesis of acute cortical necrosis. Gelfand *et al.* (1970) found lymphocytotoxic antibody in 27% of patients with acute cortical necrosis and anti-platelet antibody in 79%.

Clinical features

In those cases of acute cortical necrosis associated with abruptio placentae patient maypresent with:

- Severe lower abdominal pain
- Per vaginal bleeding
- Hypotension / Shock
- Oliguria / anuria.

Without abruptio placentae:

- Oliguria / anuria
- Infection / diarrhea / symptoms of predisposing disease.

Acute renal failure as a result of acute cortical necrosis cannot be distinguished readily from other forms of acute renal failure such as acute tubular necrosis and renal biopsy is the only sure way of making the diagnosis during life (Lauler& Schreiner, 1958).

Diagnosis

Ultrasonography

Initially shows enlarged, swollen kidneys with reduced blood flow. Cortical tissue becomesshrunken later in the course of disease (Sefczek *et al.,* 1984).

KUB X-ray

Plain X ray of the kidney shows calcification weeks or months later (Moell, 1973).

Contrast enhanced CT scanning

Contrast enhanced CT scanning is the most sensitive modality. Diagnostic features include absent opacification of the renal cortex and enhancement of sub-capsular andjuxtamedullary areas and of the medulla without excretion of contrast medium (Kleinknecht *et al.,* 1973).

DTPA renal scan

It reveals markedly diminished perfusion with delayed or no function. It is more helpful in transplant kidneys.

Kidney biopsy

Kidney biopsy provides the definitive diagnosis and prognostic information.

Unilateral cortical necrosis

Unilateral cortical necrosis may occur rarely with ureteric obstruction on the uninvolved side (Blau *et al.,* 1971). The mechanism is not clear, but experimentally, ureteric occlusion has a similar effect on the cortical necrosis found in the generalized Schwartzman reaction.

Recovery from cortical necrosis

Partial recovery of renal function has been reported and it is likely that recovery is governed by the extent of the lesion (Walls *et al.,* 1968). Schrieiner (1979) emphasized that there is high evidence of hypertension in patients who recover from cortical necrosis and Kleinknecht *et al*. (1973) found that patients who recover, may exhibit a slower decline in renal functions associated with a progressive reduction in renal mass.

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