

CLINICAL ASPECTS

Bilateral Renal Cortical Necrosis Following Binge Drinking

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Abstract — Renal cortical necrosis (RCN) is a rare cause of acute kidney injury secondary to ischemic necrosis of the renal cortex. Acute tubular necrosis after binge drinking is usually attributed to volume depletion, idiosyncratic reaction to alcohol, rhabdomyolysis or a combination with non-steroidal anti-inflammatory drugs. Binge drinking itself as a cause of RCN has not yet been reported. We report a case of a 25-year-old Asian male who developed bilateral RCN following binge drinking.

INTRODUCTION

Renal cortical necrosis (RCN) is a rare cause of acute kidney injury (AKI) secondary to ischemic necrosis of the renal cortex (Kim, 1995). Acute tubular necrosis (ATN) after binge drinking is usually attributed to volume depletion, idiosyncratic reaction to alcohol, rhabdomyolysis or a combination with non-steroidal anti-inflammatory drugs (NSAIDs) (Prakash *et al.*, 2007). Binge drinking itself as a cause of RCN has not yet been reported. We report a case of a 25-year-old Asian male who developed bilateral RCN following binge drinking.

CASE REPORT

A 25-year-old Asian male presented with a 5-day history of nausea and vomiting and a 4-day history of back pain and anuria. He had been drinking alcohol heavily every day for 2 months, with particularly heavy alcohol intake without meals over the 3 days prior to the onset of symptoms. He had consumed a total of ~30 l of soju for 2 months. Soju is a distilled liquor made from fermented potatoes and its alcohol content is 20%. He also had consumed a total of ~13 l of beer, 3 l of soju and 0.75 l of hard liquor for last 3 days. He had also not eaten proper meals for 2 months. He had no significant medical or family history and was on no medication.

He was evaluated at a local hospital before admission and underwent a non-enhanced abdominal computed tomography (CT) due to anuric renal impairment. There was no increase in urine output after administration of 3 l of normal saline, so he was transferred to our hospital. On admission, the patient was acutely ill, complaining of discomfort over the entire abdomen and back pain. The blood pressure was 130/90 mmHg, the pulse 73 beats per minute and the body temperature 36.7°C. There was bilateral tenderness of the costovertebral angle, and he had severe anuric renal impairment (urea 75 mg/dl, creatinine 12.1 mg/dl). A urinalysis showed hematuria and proteinuria. Blood count hemoglobin level was 13.6 g/dl and

white blood count was 25 200/μl. High-sensitivity C-reactive protein concentration was 29.07 mg/l, lactate dehydrogenase (LDH) was over 3000 IU/l (normal 285–540) and a clotting screen showed slightly increased values (prothrombin time international normalized ratio 1.42 and partial thromboplastin time 49.4, control 39.8). Other laboratory data included creatinine phosphokinase 21 IU/l, aspartate aminotransferase 74 IU/l, amylase 56 U/l, lipase 31 U/l and total CO₂ 19 mEq/l. Fluorescent anti-nuclear antibody, anti-neutrophil cytoplasmic antibody and cryoglobulin were all negative, as were serologic markers for viral infection and a bacterial infection culture.

An ultrasonographic study showed normal sized, unobstructed kidneys with prominent pyramids and a hypoechoic renal cortex (Fig. 1). Abdominal contrast-enhanced computed tomography (CECT) showed diffuse and bilateral cortical hypodense areas surrounded by capsular enhancement in both kidneys (Fig. 2). Hemodialysis was initiated on the day of admission. His daily urine output increased to 1500–2000 ml on the 8th day of dialysis, but his serum creatinine level did not decrease. Renal biopsy was performed on the 28th day of admission. Percutaneous kidney biopsy showed extensive coagulation necrosis in subcapsular cortex and hemorrhage with ghostly renal architecture (Fig. 3A). The central necrotic area showed a loss of normal cytologic features of necrotic tubular epithelial cells, markedly congested glomeruli (Fig. 3B), focal interstitial hemorrhage and fibrin thrombi in an interlobular artery. The histological findings of coagulation necrosis and hemorrhage are consistent with diffuse cortical necrosis. On immunofluorescence study, focal trace immunoglobulin M staining was noted in the glomeruli which tended to exclude an immune-mediated glomerulopathy. He underwent allogeneic kidney transplantation 6 months later.

DISCUSSION

RCN accounts for 2% of all cases of AKI (Kim, 1995; Prakash *et al.*, 2007). The lesions are caused by significantly diminished renal arterial perfusion secondary to vascular spasms, microvascular injury or intravascular coagulation.

The accurate pathogenesis of RCN is not known, but reported main causes include sepsis, prothrombic states, acute pancreatitis, drugs and trauma (Kim, 1995; Prakash *et al.*, 2007). Binge drinking of alcohol has not yet been reported as a direct cause of RCN.

AKI after binge drinking is usually attributed to volume depletion, a condition produced by vomiting and urinary

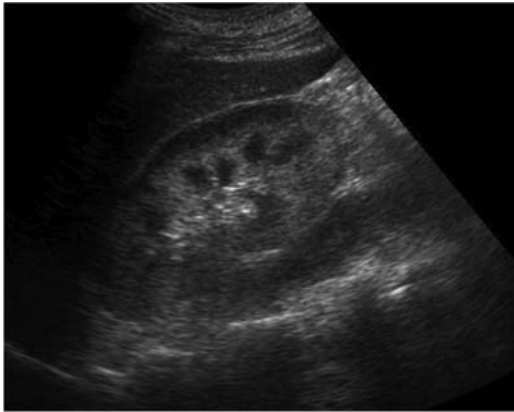


Fig. 1. Right kidney ultrasonography showed a hypoechoic renal cortex.

loss. This in turn may be caused by the suppression of antidiuretic hormone (ADH) by alcohol, an idiosyncratic reaction to alcohol or alcohol-induced rhabdomyolysis (Hirsch *et al.*, 1994). Binge drinking associated with the use of NSAIDs that interfere with renal autoregulation is a risk factor for the development of AKI (Johnson and Wen, 1995). AKI is reversible and has led to ATN in previously reported cases in which a biopsy was performed. We were able to rule out AKI due to binge drinking associated with NSAIDs because our patient had not taken any NSAIDs. We also ruled out AKI due to other causes because there was no evidence of alcohol-associated pancreatitis or rhabdomyolysis and the renal biopsy revealed RCN rather than simple ATN.

On admission to our hospital, we began dialysis because of continuing anuria after a sufficient intravascular volume load. Before dialysis, we performed an abdominal CECT to rule out RCN due to elevated LDH levels and sudden back pain. An elevated LDH level is highly sensitive for acute renal infarction and is usually not observed in patients with other types of AKI. Our patient had no risk factors for renal infarction (such as atrial fibrillation). If renal infarctions including RCN are suspected, CECT may be an appropriate diagnostic tool because RCN can be missed on non-CECT and contrast-induced kidney injury can be prevented by dialysis.

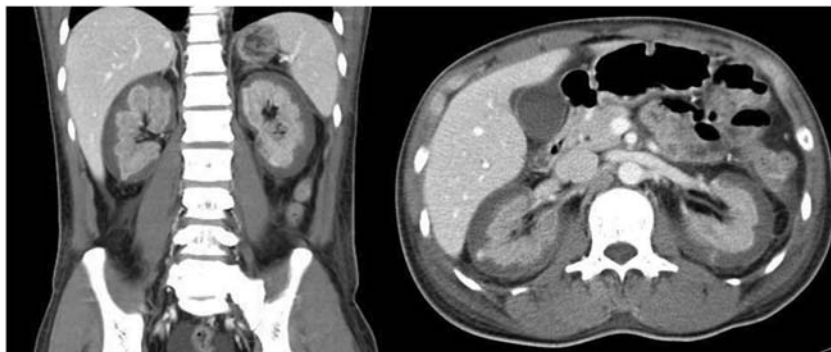


Fig. 2. Contrast-enhanced CT of kidneys. CT showed lack of renal cortical enhancement, rim subcapsular cortical enhancement and medullary enhancement in both kidneys.

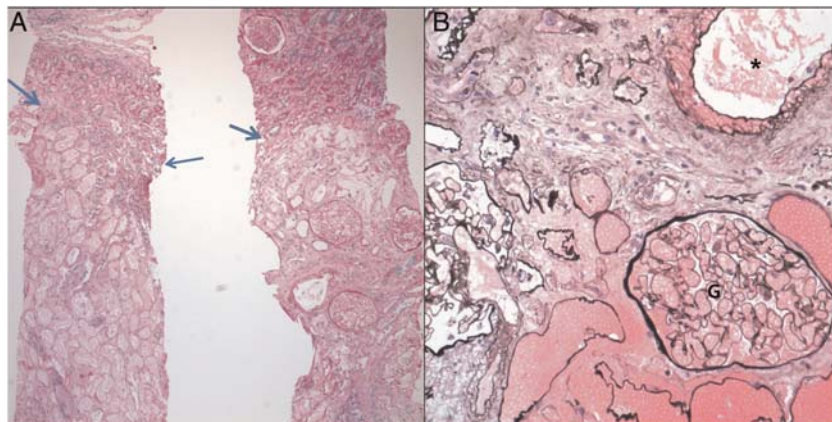


Fig. 3. Renal biopsy specimen. Diffuse coagulation necrosis of renal cortex (arrow) can be seen in both cores with relative preservation of architecture of the tubules and the glomeruli (A, periodic acid-schiff, $\times 100$). Central necrotic area shows loss of normal cytotologic feature of necrotic tubular epithelial cells and markedly congested glomeruli (G), focal interstitial hemorrhage and fibrin thrombi (*) in an interlobular artery (B, Jones methenamine silver, $\times 400$).

Lipotropic factors such as choline, methionine, B₁₂ and folic acid play important roles in diverse biological species. In rats, diets deficient in these factors can cause renal disorders such as ATN and RCN (Klatskin and Krehl, 1954; Montes *et al.*, 1980). In the kidney, choline is involved in the syntheses of phospholipids and the neurotransmitter acetylcholine and is oxidized to produce the methyl donor betaine (Parks and Smith, 1969; Acara and Rennick, 1972). Choline deficiency produces acute renal lesions in young rats, which are often associated with fatal hemorrhagic necrosis approximately 6–10 days after the implementation of a choline-deficient diet (Griffith, 1958). Alcohol increases choline requirements and may induce a state of relative deficiency when the diet is marginal in lipotropic activity (Parks and Smith, 1969). Therefore, alcohol can aggravate choline deficiency, and severe choline deficiency can cause RCN in rats. Archer *et al.* (2010) reported RCN in a fussy eater and suggested that severe hyperhomocysteinemia as a result of vitamin deficiency is associated with increased risk of endothelial activation and arterial thrombosis (Remacha *et al.*, 2002). Unfortunately, we did not check the status of lipotropic factors in our patient. However, daily alcohol consumption and binge drinking without meals can lead to vitamin deficiency, including that of choline. We suggest vitamin deficiency aggravated by binge drinking as the possible cause of RCN in our patient.

Binge drinking is not rare in the general population. Its potential nephrotoxicity (including ATN and RCN) should be brought to the attention of medical professionals as well as the general public. Vitamin deficiency is a possible cause of RCN in binge drinkers.

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