CASE REPORT

Transient Hyperechoic Renal Cortex Caused by Dehydration and Induced Acute Renal Failure in Two Patients with Intra-Abdominal Infection

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Increased renal cortical echogenicity can be seen in patients with various underlying renal abnormalities. However, there are no reports of hyperechoic cortex associated with volume depletion until now. Here, we describe two cases of hyperechoic cortex caused by severe dehydration due to liver abscess and acute salmonellosis which lead to nausea, vomiting, and diarrhea. After administering large amounts of fluid supplements, the renal functions dramatically recovered and the echogenicity of the renal cortex returned to normal. Redistribution of renal blood flow and cortical ischemia may play a role in changes in echogenicity that occur in the renal cortex. Additionally, studies on increased renal cortical echogenicity and dehydration are reviewed.

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Introduction

The echogenicity of the cortex is a crucial parameter that must be considered during sonographic evaluation of renal diseases. Increased cortical echogenicity can be seen in patients with various underlying renal abnormalities, including glomerulonephritis, chronic renal failure, drug-induced nephrotoxicity, renal cortical necrosis, etc. [1—9]. To the best of our knowledge, there are no reports on dehydration-induced hyperechoic renal cortex in the medical literature until now. In this paper, we describe two patients with hyperechoic cortex caused by dehydration. Renal cortical echogenicity returned to normal after fluid supplement.

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Case reports

Case 1

A 22-year-old male complaining of abdominal pain was admitted to the ward. He presented with anorexia, vomiting, and diarrhea for 2 weeks. He was healthy before this episode and no history of drug abuse. Upon physical examination, his blood pressure was 110/61 mmHg, his pulse rate was 126 beats/min, and his body temperature was 39.0°C. Tenderness of the right upper quadrant and poor skin turgor were noted. Laboratory data were as follows: white blood cell count, 37.1 × 10^9/L; hemoglobin concentration, 13.8 g/dL; platelet count, 166 × 10^9/L; blood urea nitrogen (BUN), 79 mg/dL; creatinine (Cr), 2.8 mg/dL; C-reactive protein, 24.1 mg/dL; albumin, 2.7 g/dL; total protein, 5.2 g/dL; creatine kinase 70 U/L; alanine transaminase, 146 IU/L; and aspartate transaminase, 59 IU/L. Peripheral blood smear was normal without any fragmented red blood cells. Urine analysis showed only an increase in specific gravity, which was measured as 1.030. The urinary fractional excretion of sodium (FE Na) was 0.75%. Abdominal computer tomography (CT) and ultrasonography revealed a huge liver abscess over the right lobe, about 14 cm in size. Ultrasonography of the kidney revealed a normal kidney size but increased echogenicity in the renal cortex equal to that of the liver. Increased corticomедullary differentiation was also found (Fig. 1). Spectral Doppler ultrasound of the interlobar artery showed a peak flow of 39 cm/sec and a resistive index (RI) of 0.71 (Fig. 1). A diagnosis of the liver abscess and prerenal azotemia due to dehydration were made.

The patient was treated with ultrasonography-guided aspiration and pig-tail catheter insertion to drain the liver abscess. Empirical antibiotics were intravenously administered initially and then according to the blood culture, which revealed Klebsiella pneumoniae. Additionally, intravenous administration of a large amount of fluid for hydration was given to treat prerenal azotemia. Renal function dramatically recovered within 1 week. The BUN level decreased to 15 mg/dL and the Cr level decreased to 0.9 mg/dL. Repeated ultrasonography showed normal kidneys and echogenicity (Fig. 2).

Unfortunately, rupture of the liver abscess into the subphrenic space, right abdomen, and pelvis occurred on the 10th day after admission. Ultrasonography-guided drainage tubes were inserted into those spaces. After this invasive procedure and the administration of antibiotic therapy, the patient continued to improve clinically and was discharged 1 month later. He had been doing well with normal renal function tests for 6 months after discharge.

Case 2

A 54-year-old male was admitted to the ward through the emergency department. He complained of the acute onset

![Fig. 1](https://example.com/fig1.png)  
Ultrasonographic images of the liver and kidneys on admission. (A) A large abscess in the right lobe of the liver is noted, which was composed of echogenic parts (arrows) and liquefied fluid spaces (arrowhead). (B,C) Bilateral kidneys showing increased renal cortical echogenicity with increased corticomedullary differentiation (arrows). (D) Spectral Doppler study showing a peak flow of 39 cm/sec in the interlobar artery with a resistivity index of 0.71.
of increased frequency of bowel movements (up to 20 times per day) with watery and mucin-containing diarrhea for 2 days. He appeared to be chronically ill with the loss of skin turgor. He presented with nausea, vomiting, and acute abdominal cramps followed by chills, myalgias, and watery diarrhea. Physical examination revealed mild fever (37.5°C) and severe prostration. Mild tenderness was noted in the periumbilical region. His blood pressure was 86/40 mmHg and his pulse rate was 122 beats/minute. Laboratory data were as follows: while blood cell count, 12.2 x 10^9/L; hemoglobin concentration, 13.2 g/dL; BUN, 64 mg/dL; Cr, 2.1 mg/dL; C-reactive protein, 13.5 mg/dL; albumin, 3.1 g/dL; total protein, 5.1 g/dL; creatine kinase, 46 U/L; alanine transaminase, 52 IU/L; and aspartate transaminase, 49 IU/L. An abdominal ultrasonography performed before admission showed mild fatty infiltration of the liver and unusually increased echogenicity of the renal cortex in both kidneys (Fig. 3A). The right kidney measured 10.6 cm and the left kidney measured 10.9 cm in the craniocaudal dimension. Spectral Doppler sonography was not performed. Under the impression of acute bacterial gastroenteritis or acute bacterial food poisoning related to salmonellosis, patient blood samples were collected and sent for culturing. The available food that was eaten 2 days prior to the onset of symptoms and stool samples were also sent for culturing. Four hours after admission, broad-spectrum antibiotics were intravenously administrated. A large amount of fluids for hydration was also intravenously administered. Renal function recovered within 3 days and ultrasonography 2 days after intravenous hydration and antibiotic administration demonstrated normal renal echogenicity (Fig. 3B). Five days after medical treatment, the results from the cultures of blood and available offending food revealed *Salmonella*, confirming the initial clinical impression. The patient was treated accordingly and was discharged 3 days after the total disappearance of his clinical symptoms. He was doing well at least 4 months after discharge.

**Discussion**

Renal cortical echogenicity varies with age. After 6 months of age, a normal renal cortex is never more echogenic than the liver, and thereafter echogenicity progressively decreases with age [1]. Glomeruli and tubules are the principle interfaces that contribute to the echogenicity of the normal renal cortex [2]. In patients with renal diseases, tubular atrophy and interstitial inflammation are significant determinants of cortical echogenicity [3]. Disorders associated with hyperechoic cortex include the following: certain infiltrative diseases [4], glomerulonephritis, chronic renal failure [5], drug-induced nephrotoxicity [6], renal cortical necrosis [7], renal injury due to perinatal hypoxia [8], and exposure to aliphatic solvents [9]. In our patients, judging from their history of dehydration, poor skin turgor, increased urinary specific gravity, BUN/Cr ratio, and FENa, a prerenal cause of renal failure was favored. Although one of them presented with concomitant liver abscess-
associated sepsis, sepsis-related acute tubular necrosis could be excluded by the resistive index of 0.71 [10]. The hyperechoic cortex was caused by fluid volume depletion, which was confirmed by the dramatic recovery of renal function and the return of renal cortical echogenicity after a large amount of fluid supplement.

Despite the frequency of volume depletion-related renal failure, there have been very few relevant studies that correlate volume depletion and renal cortical echogenicity. The definitive mechanism is unknown, however, circulatory redistribution might play a role in the phenomenon of renal cortical hyperechogenicity [8]. Burgener et al reported a physiological redistribution of blood flow away from the cortex to the medulla in dehydrated normotensive dogs. Additionally, an increased incidence of cortical ischemia, which was readily reversible with an infusion of isotonic saline, was also noted [11]. Hypoxemia in newborns can also cause renal cortical hyperechogenicity. This finding is most likely related to changes in vascular perfusion that occur in the renal cortex because perinatal hypoxia is generally associated with hypovolemia [12]. Tovbin et al also reported the case of a hyperechogenic right kidney due to unilateral acute renal cortical necrosis following skipping with a rope; selective right renal angiography showed no filling of the interlobular or arcuate arteries [13].

Ultrasonography is an important tool for diagnosing kidney diseases. Increased renal cortical echogenicity is a nonspecific but significant finding that suggests the presence of various underlying renal abnormalities. In this report, we describe the first case of hyperechoic renal cortex caused by dehydration in a patient with liver abscess. Temporary physiological redistribution of renal blood flow and cortical ischemia may play a role in the disease mechanism. However, the mechanism behind this sonographic phenomenon and volume depletion requires further investigation.

References