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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.

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 corticomedullary 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 administrated 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 occured 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

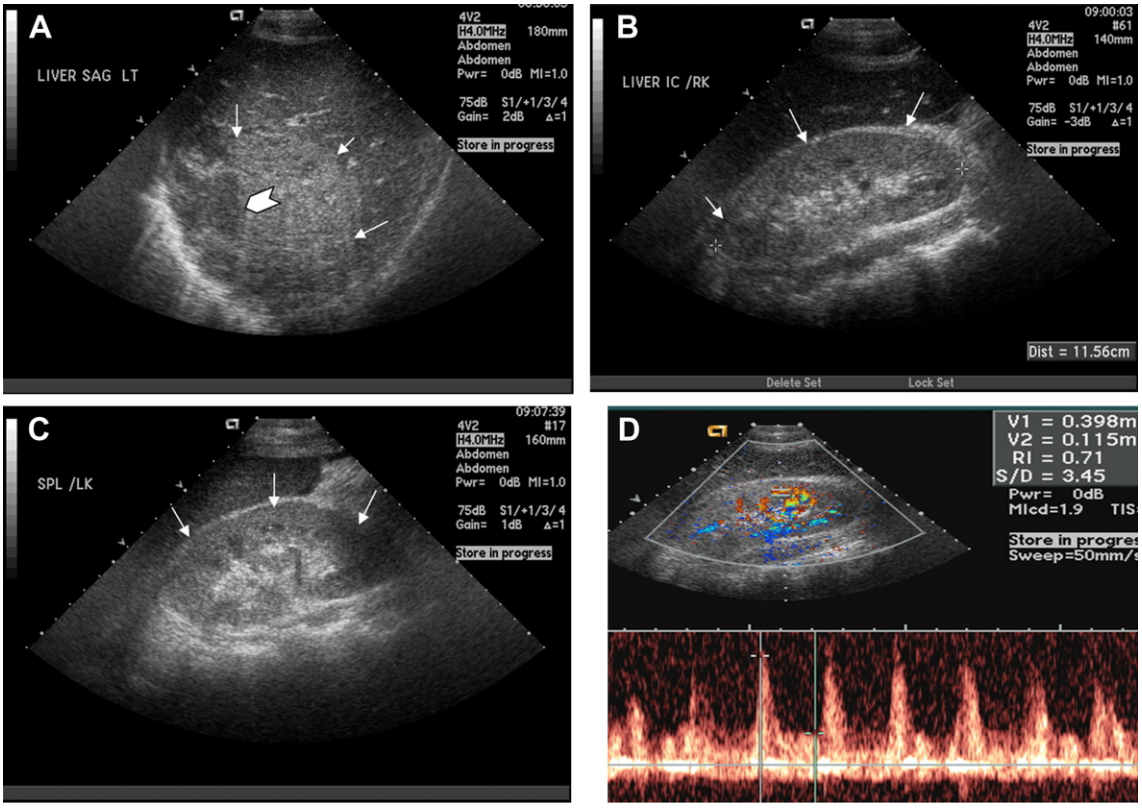

Fig. 1 Ultrasonographic images of the liver and kidneys on admission. (A) A large abscess in the right lobe of the liver is noted, which was composed of echogenic parts (arrows) and liquefied fluid spaces (arrowhead). (B,C) Bilateral kidneys showing increased renal cortical echogenicity with increased 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.