Increased echogenicity of renal cortex: a transient feature in acutely ill children.

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Abstract

Introduction:
The purpose of our study was to determine the frequency of hyperechogenicity of renal parenchyma in children with acute abdominal illness and to evaluate the assumed transient feature of this hyperechogenicity.

Materials and methods:
Between January 2005 and February 2006, 189 consecutive patients (112 boys and 77 girls; mean age, 10 years) presenting with acute abdominal pain were examined with ultrasonography. Patients with a known history of renal disease and those with acute urinary tract infection were excluded from the study. Echogenicity of renal cortex in comparison with adjacent liver was recorded. Renal cortex echogenicity was divided into three groups; group 1, renal cortex echogenicity less than liver parenchyma echogenicity; group 2, renal cortex echogenicity similar to that of liver parenchyma; and group 3, renal cortex echogenicity greater than that of liver parenchyma. Patients with hyperechogenicity were reexamined with ultrasonography after 2 weeks or more. The final ultrasonographic diagnosis and clinical outcome were noted.

Results:
Renal cortex echogenicity was equal to or greater than that of liver parenchyma in 18% (n = 34) of 189 patients. Increased echogenicity of the renal cortex returned to normal in two or more weeks time in all patients. Three patients had no follow-up. Clinical diagnoses were idiopathic acute abdominal pain (n = 74), appendicitis (n = 83), mesenteric lymphadenitis (n = 15), ileoceccit (n = 7), gastroenteritis (n = 7), Crohn disease (n = 1), intussusception (n = 1), and pneumonia (n = 1). No concurrent renal disease was diagnosed.

Conclusion:
Increased echogenicity of renal parenchyma in children with acute illness is a transient feature and does not necessarily indicate renal disease.
Introduction

Ultrasonography (US) is an important diagnostic tool in the evaluation of acute abdominal illness in pediatric patients [1, 2]. In most cases, patients do not have an established diagnosis at the time of the ultrasonographic examination. The clinical differential diagnosis is extensive. Evaluation of the kidneys is a routine part of abdominal US. The normal renal cortex in older infants, children, and adults should be less echogenic than the liver parenchyma [3-7]. Only in neonates and infants is increased echogenicity of the renal parenchyma a normal finding [6]. Increased renal echogenicity of infants changes gradually in the adult pattern [7]. Several reports have stated that increased echogenicity of the renal parenchyma is an indicator of renal disease [3, 4]. However, we have encountered increased echogenicity in patients with various abdominal diseases but no concurrent renal disease.

The purpose of this study was to determine the frequency of hyperechogenicity of the renal cortex in children with acute abdominal illness, but no history of renal abnormalities, and to assess the assumed transient feature of this finding.

Materials and methods

Patients

From January 2005 to February 2006, all consecutive pediatric patients with acute abdominal illness (acute abdominal pain of < 8 days, with or without nausea, fever, vomiting, or diarrhea) were referred by the pediatric surgeon or a pediatrician of the emergency department to the department of radiology for an abdominal ultrasonographic examination. None of the patients was resuscitated before the examination. Only one patient was not hemodynamically stable during the examination. No medication was given before the ultrasonographic examination. All patients with a history of renal disease (including recent acute pyelonephritis or glomerulonephritis, acute fungal infections, stasis nephropathy, hemolytic-uremic syndrome and renal artery or renal vein thrombosis), renal neoplasms, kidney transplantation, solitary kidney or other renal anomalies, multicystic dysplastic kidney disease, urologic surgery or vesicoureteral reflux were excluded from the study (n = 4). Patients with concurrent use of diuretics were excluded. All patients younger than two years old were also excluded from the study (n = 8) because the renal cortex in normal neonates and infants can be hyperechoic [6, 7]. Of a total of 201 patients with acute abdominal illness who where referred for abdominal US, 189 patients with a mean age of 10 years (age range, 2-15 years) were included in our study. Final diagnoses after US and after additional US or CT if necessary as well as findings of clinical or pathological evaluation were noted. Serum creatinine was tested and urine analysis performed for 17 and 23 patients, respectively, with increased renal cortex echogenicity. After 1 year, records of patients with increased echogenicity of the renal parenchyma records were evaluated for renal disease.
Informed consent was obtained from each patient or his or her parents in conformance with the rules of our country. Institutional review board approval was obtained for our observational study.

Imaging observations
All children underwent comprehensive abdominal US. The images were obtained by using a HDI 5000 scanner (ATL HDI 5000; Philips Medical Systems) with a curved array (2-5 MHz) transducer. The ultrasonographic examinations were performed by a pediatric radiologist, a staff radiologist, or a resident in radiology. The radiologists had 6-14 years of experience in pediatric abdominal US. The residents were in the third or fourth year of their medical training and had about 6 months of specific experience.

Transverse and longitudinal images of the kidneys were obtained. The renal parenchyma was analyzed for diffuse renal cortex echogenicity. Renal cortex echogenicity of the right kidney was compared with echogenicity of the liver parenchyma and classified into three groups: group 1, in which renal cortex echogenicity was less than liver echogenicity; group 2, in which renal cortex echogenicity was equal to liver echogenicity; and group 3, in which renal cortex echogenicity was greater than liver echogenicity. Renal cortex echogenicity was determined by the radiologist during the examination.

Almost all patients with increased renal cortex echogenicity (i.e., patients in groups 2 and 3) were reexamined after 2 weeks. At the time of reexamination, the acute abdominal complaints had resolved (range until reexamination, 1 day-1 year). Three patients were not reexamined.

Statistical analysis
The chi-square test was used for the comparison of subgroups. A p value of less than 0.05 was considered to indicate a statistical difference.

Results
General Characteristics
Final ultrasonographic diagnoses in the 189 included patients were normal findings (n = 74), appendicitis (n = 84), mesenteric lymphadenitis (n = 15), ileocecalitis (n = 7), bowel wall thickening (as in colitis, Crohn disease and the clinical diagnosis of infectious gastroenteritis) (n = 8) and intussusception (n = 1). No concurrent renal disease or urinary tract infection was diagnosed in the study population. Table 1 shows the ultrasonographic diagnosis and the final clinical diagnosis (after the ultrasonographic examination and, if necessary, additional chest radiography, US, or CT, as well as clinical and pathological results). Serum creatinine and urine sediment were normal in all tested patients in groups 2 and 3. None of the patients with increased renal cortex echogenicity had a renal disorder after 1 year of clinical follow-up.
**Table 1** Ultrasonographic and final clinical diagnoses in 189 children who underwent abdominal US

<table>
<thead>
<tr>
<th>Finding</th>
<th>Ultrasonographic diagnosis</th>
<th>Clinical diagnosisa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal findings</td>
<td>74</td>
<td>NA</td>
</tr>
<tr>
<td>Idiopathic abdominal pain</td>
<td>NA</td>
<td>74</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>84</td>
<td>83</td>
</tr>
<tr>
<td>Mesenteric adenitis</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Ileocecitis</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Thickening of bowel wallc</td>
<td>8</td>
<td>NA</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>NA</td>
<td>7</td>
</tr>
<tr>
<td>Crohn disease</td>
<td>NA</td>
<td>1</td>
</tr>
<tr>
<td>Intussusception</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>NA</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>189</td>
<td>189</td>
</tr>
</tbody>
</table>

Note—NA= not applicable

a Based on radiological, clinical and pathological results.
b One patient with positive US findings of appendicitis underwent negative appendectomy, clinical diagnosis was idiopathic abdominal pain.
c As seen in colitis, Crohn disease, and in patients with the clinical diagnosis gastroenteritis.

**Imaging findings**

In 34 (18%) of the 189 patients, echogenicity of the renal cortex was increased in comparison with that of the adjacent liver. In 15 (8%) patients, the renal echogenicity was equal to the liver (group 2), and in 19 (10%) patients renal echogenicity was greater than the liver (group 3). Figures 1 and 2a show the hyperechogenicity of the renal cortex in two patients with appendicitis. Renal cortex echogenicity was increased in patients with appendicitis, gastroenteritis, mesenteric lymphadenitis, ileocecitis, Crohn disease and pneumonia. Table 2 shows the type of recorded renal cortex echogenicity and the final clinical diagnosis of all patients. Clinical diagnosis was based on ultrasonographic, clinical and pathological results.

In the subgroup \( n = 83 \) with appendicitis, the echogenicity of the renal cortex was equal to liver parenchyma in 12 cases, and in 14 cases the renal cortex echogenicity was more than the liver parenchyma in children with appendicitis. Prevalence of appendicitis in this study population was 44% (83/189). In total, 26 (31%) of the 83 patients with appendicitis had hyperechogenicity of the renal cortex. Three of these 26 patients had a perforated appendix or an appendicular abscess. Renal cortex echogenicity returned to normal in all reexamined patients, as shown in Figure 2b. In one patient, the increased echogenicity returned to normal in 1 day, detected coincidentally during screening for persistent pain after surgical resection of an acutely inflamed appendix.
Table 2 Renal cortex echogenicity correlated with clinical diagnosis

<table>
<thead>
<tr>
<th>Clinical diagnoses</th>
<th>Renal cortex echogenicity</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Less than liver</td>
<td>Equal to liver</td>
<td>Greater than liver</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Idiopathic abdominal pain</td>
<td>73</td>
<td>0</td>
<td>1</td>
<td>74</td>
<td></td>
</tr>
<tr>
<td>Appendicitis</td>
<td>57</td>
<td>12</td>
<td>14</td>
<td>83</td>
<td></td>
</tr>
<tr>
<td>Mesenteric adenitis</td>
<td>14</td>
<td>0</td>
<td>1</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Ileocecitis</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Crohn disease</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Intussusception</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>155</strong></td>
<td><strong>15</strong></td>
<td><strong>19</strong></td>
<td><strong>189</strong></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1.
10 year-old boy with appendicitis. Ultrasound of right kidney in longitudinal plane shows renal cortex echogenicity equal to that of liver parenchyma.
Increased echogenicity of renal cortex: a transient feature in acutely ill children

Figure 2. 13 year old girl with appendicitis. Ultrasound (a) shows renal cortex echogenicity is higher than that of liver parenchyma. After appendectomy, follow-up ultrasound (b) shows renal cortex echogenicity has returned to normal.

Statistical analysis
Renal cortex echogenicity was not significantly \( (p = 0.66) \) greater in patients with a perforated appendix or a periappendicular abscess than in patients with uncomplicated appendicitis. However, renal cortex echogenicity was statistically significantly \( (p = 0.000033) \) greater in children with appendicitis (both uncomplicated and complicated) than in children with abdominal pain not diagnosed as appendicitis.
Discussion

In this study of 189 children with acute abdominal pain, 34 (18%) had increased echogenicity of the renal cortex. In contrast to previous studies [4, 8, 9], we did not find this hyperechogenicity to be an indicator of renal disease in this specific patient population. None of these patients had a history of renal disease, nor did they develop a renal disorder after one year follow-up. Because previous articles [3, 5] have shown that in healthy pediatric subjects (neonates and infants excluded) echogenicity of the renal parenchyma is not increased, we believe that hyperechogenicity of renal parenchyma can be a nonspecific finding, a sign of (abdominal) illness varying from appendicitis to ileoceleitis, infectious gastroenteritis or otherwise, rather than an indicator of renal disease. Only one study reported that renal echogenicity equal to the liver parenchyma is a nonspecific finding and does not suggest renal disease; that study, however, included only adults [10].

The cause of increased renal cortex echogenicity remains unclear. Manley and O’Neill [11], believed that renal cortex echogenicity can be influenced by the state of diuresis or hydration. Those authors reported that echogenicity is greater in well-hydrated healthy patients. Although we have no proof, we believe, contrary to Manley and O’Neill, that increased renal cortex echogenicity can be explained by a less hydrated state of the patients rather than a more hydrated state. In our study, patients tended to have decreased hydration as a consequence of their acute abdominal illness due to vomiting, diarrhea, and decreased intake. An explanation might be the increase of acoustic interfaces as a consequence of swelling of interstitial renal parenchyma. However, this is not evidence-based. Unfortunately, routine evaluation of blood parameters in the emergency department includes no electrolyte evaluation or serum creatinine, urea or liver function parameters. However, serum creatinine was normal in all tested cases. This finding is in accordance with the findings of Kasap et al. [9], who found no significant difference in blood parameters (serum creatinine) in children with a renal cortex echogenicity equal to that of the liver or in those children with renal cortex echogenicity more than that of the liver parenchyma.

A limitation of this study was the subjectivity of the evaluation of the cortical echogenicity. Few reports have stated that densitometric measurement of echogenicity is more reliable than naked eye evaluations [10, 12, 13]. However, in our institute these measurements are not made in regular practice.

Another limitation was that the echogenicity of liver parenchyma was used as an internal standard in determining renal cortex echogenicity. Changes in the liver could influence the ratio of the renal cortex echogenicity in the absence of changes in the structure of the kidney. No studies are available that support a change in liver echotexture in acutely ill children. In a study on hepatic echogenicity, however, Samad et al. [14] detected no statistically significant difference in echogenicity of the liver in children with hepatic dysfunction and children in a control group. We think, therefore, that comparison with liver parenchyma can be used as a reliable method.
According to most studies of echogenicity of the renal parenchyma, right kidney echogenicity is best compared with the echogenicity of the adjacent liver [3, 11, 13, 14]. Care should always been taken, however, in interpreting the estimates of renal cortex echogenicity [15]. According to Vehmas and Kaukiainen [16], the optimum method for evaluating renal cortex echogenicity in daily practice remains to be clarified.

This study was limited by lack of a control group. At the outset, we thought that a control group would be redundant because previous studies had already proven that renal cortex echogenicity in normal healthy patients is less than that of the adjacent liver parenchyma [3, 5].

In conclusion, the results of this study show that increased echogenicity of the renal cortex in children with acute abdominal illness is a nonspecific finding and does not necessarily indicate true renal disease. Increased echogenicity of the renal cortex is a transient phenomenon in this clinical setting. Hyperechogenicity of the renal cortex in children with acute abdominal illness should alert the radiologist to search the abdomen more thoroughly for a cause of the acute abdominal illness, such as appendicitis.
Chapter 4

References