Case Reports in Gastroenterology

Case Rep Gastroenterol 2016;10:452–458

DOI: 10.1159/000446765
Published online: August 31, 2016

Case Report

Signal Intensity of Superb Microvascular Imaging Correlates with the Severity of Acute Cholecystitis

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Keywords
Color-coded superb microvascular imaging · Monochrome superb microvascular imaging · Acute cholecystitis · Percutaneous transhepatic gallbladder drainage

Abstract
Evaluation of the severity of acute cholecystitis is critical for the management of this condition. Superb microvascular imaging (SMI) enables the assessment of slow blood flow of small vessels without any contrast medium. An 84-year-old man visited our hospital with right upper abdominal pain. Computed tomography and abdominal ultrasonography showed a slight thickening of the gallbladder. White blood cell count and C-reactive protein levels were elevated. He was diagnosed with acute cholecystitis and treated conservatively with antibiotics. Two days later, his condition worsened and percutaneous transhepatic gallbladder drainage (PTGBD) was performed. The patient recovered and was discharged, and his drainage was withdrawn 7 days later. On admission, color-coded SMI (cSMI) showed pulsatory signals

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DOI: 10.1159/000446765

on the slightly thickened gallbladder wall. On the day of PTGBD, the intensity of the signal on cSMI had increased. Once the patient was cured, no further signal was observed on the gallbladder wall with either cSMI or mSMI. In conclusion, the strong pulsatory signal correlated with the severity of acute cholecystitis observed with cSMI and mSMI. Illustrating the signal intensity is useful for the evaluation of the severity of acute cholecystitis.

Introduction

Acute cholecystitis is an inflammation of the gallbladder due to obstruction of the neck or cystic duct, primarily caused by gallstones [1]. Acute cholecystitis is characterized as a combination of local and systemic inflammation [2], and its treatment is dependent on the severity [3], although it is often treated conservatively with antibiotics [4]. When acute cholecystitis becomes severe, it is treated surgically [5], while for some patients, percutaneous transhepatic gallbladder drainage (PTGBD) is an alternative to surgery [6]. An accurate assessment of the severity is critical for the appropriate management of acute cholecystitis.

Abdominal ultrasonography (US) is useful for the diagnosis of diseases of the abdominal cavity [7]. With abdominal US, acute cholecystitis shows distension, wall thickening, or sludge in the gallbladder [2]. Evaluation of the severity is based on organ failure and blood test variables [8]. However, this condition can be treated more appropriately if the severity is evaluated, using diagnostic imaging prior to the occurrence of organ failure or worsening of blood test variables. With regard to diagnostic imaging, wall thickening is representative of edema and correlates with the severity of acute cholecystitis [9], while inflammation is indicated by both edema and increased blood flow. If blood flow is assessed using abdominal US, it is possible to more accurately evaluate the severity of acute cholecystitis.

Superb microvascular imaging (SMI) is based on color Doppler imaging of abdominal US and enables the visualization of very slow blood flow without requiring contrast medium [10]. SMI utilizes an algorithm directed at small blood vessels with slow flow velocity. It is composed of color-coded (cSMI) and monochrome SMI (mSMI), where cSMI visualizes pulsatory signals such as color Doppler images and mSMI enhances the blood flow signal by suppressing background signaling. Both types of SMI are more sensitive than color and power Doppler imaging [11].

Here, we present the changes observed using cSMI and mSMI over the course of an acute cholecystitis in 1 patient.

Case Presentation

An 84-year-old man was admitted to the National Hospital Organization Shimoshizu Hospital with right upper abdominal pain that persisted for 3 days. His blood test variables are shown in table 1. White blood cell count (WBC) and C-reactive protein (CRP) levels were $11.4 \times 10^3/\mu l$ and 15.3 mg/dl, respectively. These results suggested inflammation. Total bilirubin, alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, and γ-glutamyl transpeptidase levels were 1.5 mg/dl, 206 IU/l, 20 IU/l, 10 IU/l, and 10 IU/l, respectively. The possibility of obstructive jaundice was low. To clarify the cause of inflammation, computed tomography (CT) scans (SOMATOM Emotion 16; Siemens, Munich, Germany) were performed, which showed slight thickening of the gallbladder wall (fig. 1a). To confirm

the results of the CT scans, the patient was subjected to abdominal US (Applio 400; Toshiba Medical, Ohtawara, Japan) with a curved array transducer (PVT-375BT) at 3.75 MHz (Toshiba Medical). The abdominal US showed distention and thickening of the gallbladder wall (fig. 1b), and a sonographic Murphy sign was observed. These findings indicated acute cholecystitis. The severity was classified as 'mild' as the patient showed no signs of organ failure [8]. Diagnostic imaging found no evidence of marked local inflammation such as pericholecystic or hepatic abscess. The patient was treated conservatively with antibiotics (cefazolin sodium 3 g/day) intravenously from the date of admission. After 2 days of treatment, right upper abdominal pain still persisted, and his body temperature rose to 38.7°C. WBC and CRP levels rose to 15.6 × 10^3/μl and 19.6 mg/dl, respectively. These data indicated that the acute cholecystitis had worsened. To evaluate the acute cholecystitis morphologically, abdominal US was performed. Abdominal US showed that the wall thickness of the gallbladder had increased and a sonolucent area was visible (fig. 1d). Complications, such as liver abscess or peritonitis, were considered possible. To avoid complications and promote recovery, PTGBD was performed (fig. 1c). On the day after PTGBD, right upper abdominal pain had ceased and the body temperature dropped to 36.3°C. These signs suggested that PTGBD had been effective and the patient was recovering. Seven days after PTGBD, WBC and CRP levels were 6.8 × 10^3/μl and 1.2 mg/dl, respectively, also confirming recovery. Thus, cefazolin sodium was stopped 7 days after PTGBD. Fourteen days after PTGBD, the patient was discharged from our hospital. PTGBD was withdrawn a further 7 days after discharge.

On the day of admission, cSMI showed a pulsatory signal on the wall of the gallbladder with the curved array transducer (2.0 cm/s; PVT-375BT) at 3.75 MHz (fig. 2a), whereas no such signal was observed on the wall with mSMI (2.2 cm/s) (fig. 2b). On the second day of admission, when his acute cholecystitis had worsened, this signal became more evident with cSMI (fig. 2c), while mSMI showed signals on the thickened wall (fig. 2d). After PTGBD, the gallbladder shrank due to drainage of the gall and the pulsatory signal was no longer visible with either cSMI (fig. 2e) or mSMI (fig. 2f).

Discussion

Abdominal US is recommended as a first-line diagnostic imaging tool for the diagnosis of acute cholecystitis [4]; however, false negatives are a known issue of this technique [12]. In our case, a pulsatory signal was observed on the wall of the gallbladder on the day of admission, which suggested positive cSMI, even when wall thickening was minimal. SMI is expected to reduce the incidence of false negatives when diagnosing this condition. In the present case, the strength of the pulsatory signals correlated with the severity of the gallbladder inflammation, as illustrated using both cSMI and mSMI. The results suggest that the signal intensity of cSMI and mSMI is correlated with the severity of inflammation. Our case shows that both cSMI and mSMI are useful for the diagnosis and evaluation of the severity of acute cholecystitis.

Contrast-enhanced abdominal US shows a strong enhancement that successfully differentiates acute cholecystitis from a healthy gallbladder and chronic cholecystitis [13]. However, unlike contrast-enhanced abdominal US, SMI does not require contrast medium and is therefore suitable for the management of acute cholecystitis.

A primary limitation of the present study is that it is based on just 1 case of acute cholecystitis. In order to confirm the usefulness of SMI for the evaluation of the severity of acute cholecystitis, more patients should be enrolled in larger studies and SMI findings for acute

and chronic cholecystitis compared. Furthermore, signal intensity should be stratified against the severity of acute cholecystitis.

In conclusion, a strong pulsatory signal correlated with the severity of acute cholecystitis with both cSMI and mSMI and may be suitable for the evaluation of the severity of this condition.

Statement of Ethics

This report was approved by the Ethical Committee of the National Hospital Organization of Shimoshizu Hospital. The study was considered an element of daily clinical practice as opposed to a clinical trial. Written informed consent was obtained for the performance of contrast-enhanced CT and PTGBD. The patient's records were anonymously and retrospectively analyzed, and written informed consent was obtained for their use in this report.

Disclosure Statement

The authors have no conflicts of interest to declare.

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DOI: 10.1159/000446765

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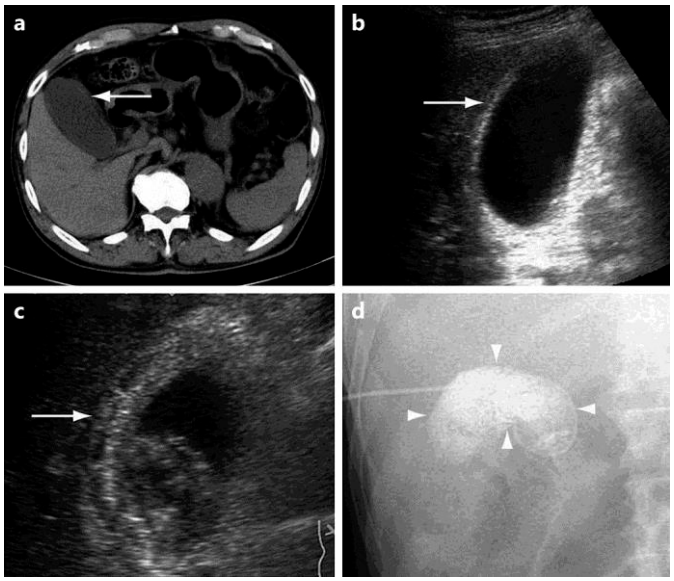

Fig. 1. Diagnostic imaging of acute cholecystitis. **a** CT shows wall thickening of the gallbladder (arrow). **b** Abdominal US shows distention and slight wall thickening of the gallbladder and sonographic Murphy sign (arrow). **c** Two days after treatment with antibiotics, the wall thickness had increased and a sonolucent area appeared on the wall of the gallbladder (arrow). **d** PTGBD revealed the internal space of the gallbladder (arrowheads).

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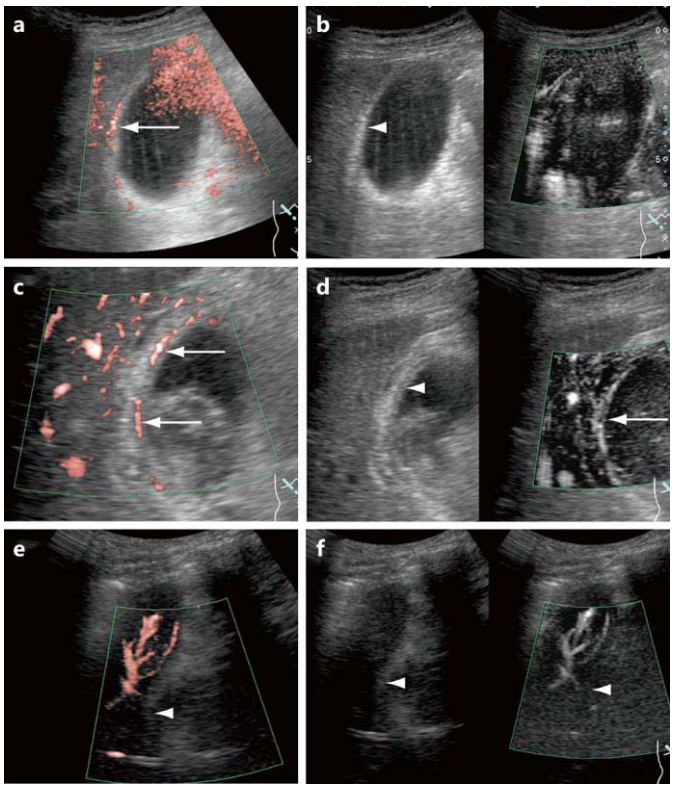

Fig. 2. SMI of acute cholecystitis. **a** On the day of admission, cSMI showed a pulsatory signal on the wall of the gallbladder (arrow). **b** mSMI did not show a significant signal on the wall of the gallbladder (arrowhead). **c** Two days later, strong pulsatory signals were observed with cSMI (arrows). **d** mSMI showed signals on the wall (arrowhead and arrow). **e**, **f** After percutaneous gallbladder drainage, no signals were observed with either cSMI (arrowhead; **e**) or mSMI (arrowheads; **f**).

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Table 1. Blood test variables

	Normal range	Days after admission			
		0	2 (PTGBD)	9	16
WBC, $\times 10^3/\mu l$	3.5–8.5	11.4	15.6	6.8	6.3
Hb, g/dl	13.5–17.0	11.5	11.8	11.2	11.3
Plt, $\times 10^4/\mu l$	15–35	13.9	15.0	23.5	26.2
CRP, mg/dl	0.0–0.3	15.3	19.6	1.2	1.0
T-Bil, g/dl	0.3–1.2	1.5	0.7	0.5	0.4
ALP, IU/l	115–359	206	270	198	231
AST, IU/l	13–33	20	16	18	14
ALT, IU/l	8–42	10	10	9	8
γ-GTP, IU/l	10–47	10	10	11	11

Hb = Hemoglobin; Plt = platelets; T-Bil = total bilirubin; ALP = alkaline phosphatase; AST = aspartate aminotransferase; ALT = alanine aminotransferase; γ-GTP = γ-glutamyl transpeptidase.