Comparison of the clinical characteristics and imaging findings of acute cholangitis with and without biliary dilatation

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Objective: To evaluate the causes of acute cholangitis without biliary dilatation and to compare the clinical characteristics and the imaging findings between patients with acute cholangitis with and without biliary dilatation.

Methods: 93 patients diagnosed with acute cholangitis underwent contrast-enhanced CT. Among them, 17 patients were classified as not having biliary dilatation (Group 1) and 76 patients were classified as having biliary dilatation (Group 2). The causes of acute cholangitis were evaluated in both groups. Clinical characteristics and imaging findings were compared between the two groups.

Results: The causes of acute cholangitis without biliary dilatation included common bile duct (CBD) stones ($n=11$), CBD sludge ($n=3$), a passed stone ($n=1$) and unknown causes ($n=2$). The total bilirubin levels of Group 1 were significantly lower than those of Group 2 ($p=0.001$). By contrast, Group 1 had higher median alanine aminotransferase (ALT) levels than Group 2 ($p=0.04$). The length of hospital stay was significantly longer in Group 2 than in Group 1 patients ($p<0.001$). In the imaging findings, the extent of transient hepatic attenuation differences (THADs) ($p=0.003$) were significantly smaller in Group 1 than in Group 2.

Conclusion: CBD stones and sludge were the most common causes of acute cholangitis in patients without biliary dilatation. These patients showed lower levels of bilirubin and higher levels of ALT than those with acute cholangitis with biliary dilatation, and had a shorter duration of hospital stay. The extent of THADs was the only discriminative CT finding between the two groups.

Advances in knowledge: Acute cholangitis can present without biliary dilatation on imaging, and the most common causes are CBD stones and sludge. The patients with acute cholangitis without biliary dilatation have different clinical characteristics and imaging findings compared with those with acute cholangitis presenting with biliary dilatation.

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In patients with acute cholangitis, an increase in intraductal pressure results from biliary obstruction. An intraductal pressure level high enough to cause cholangiovenous or cholangiolymphatic reflux may result in the systemic inflammatory response syndrome [1].

One of the diagnostic criteria for a definite diagnosis of acute cholangitis is the presence of Charcot's triad. In patients without all the components of Charcot's triad, laboratory data and imaging findings are used to support the clinical diagnosis, based on the guidelines proposed by members of the International Consensus Meeting held in Tokyo in 2006 [1].

The imaging findings of acute cholangitis include biliary dilatation, thickening of the bile duct wall, papillitis and transient hepatic attenuation differences (THADs) [2–6]. Most patients with acute cholangitis have dilatation of the bile duct visible by imaging. However, in some patients diagnosed with acute cholangitis, dilatation of the bile duct is not present. Raper et al [7] reported that the patient group

with bile duct obstruction had a significantly higher intraductal pressure than the non-obstructed group, and demonstrated that cholangiovenous reflux of biliary bacteria increased as intraductal pressure increased. Patients diagnosed with acute cholangitis with biliary dilatation are therefore likely to have higher intraductal pressure and more frequent cholangiovenous reflux than those without biliary dilatation; subsequently, we would expect to observe a more severe form of acute cholangitis in acute cholangitis patients with biliary dilatation. However, no comparison of the clinical and imaging characteristics of patients with acute cholangitis with and without biliary dilatation has been performed prior to the current study.

Our purpose in this study was therefore to evaluate the causes of acute cholangitis without biliary dilatation and to compare the clinical characteristics and the imaging findings between acute cholangitis patients with and without biliary dilatation.

Methods and materials

The institutional review board approved this retrospective study and the requirement for informed consent was waived.

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Table 1. Diagnostic criteria for acute cholangitis (Tokyo Guidelines [1])

A. Clinical context and clinical manifestations	1. History of biliary disease
	2. Fever and/or chills
	3. Jaundice
	4. Abdominal pain (right upper quadrant or upper abdominal)
B. Laboratory data	5. Evidence of inflammatory response ^a
	6. Abnormal liver function tests ^b
C. Imaging findings	7. Biliary dilatation, or evidence of an aetiology
<i>Definite diagnosis</i>	(1) Charcot's triad (2+3+4)
	(2) Two or more items in A+both items in B+item C

^aAbnormal white blood cell count, increase in serum C-reactive protein level and other changes indicating inflammation.

^bIncreased serum alkaline phosphatase, γ -glutamyltransferase, aspartate aminotransferase or alanine aminotransferase levels.

Patient selection

Between January 2009 and December 2009, 6317 patients underwent 9364 contrast-enhanced dynamic CT scans of the abdomen. After reviewing the medical records and imaging findings of these 6317 patients, two radiologists selected 100 patients with acute cholangitis based on the diagnostic criteria established in the Tokyo guidelines (Table 1). 35 patients had all of the components of Charcot's triad and 65 patients without all the components of Charcot's triad had laboratory results indicating inflammation and abnormal liver function, with imaging findings suggestive of biliary obstruction. Seven patients were excluded from the study because they underwent CT examination more than 3 days after admission. The final study population included 93 patients comprising 59 males and 34 females (mean age 68 years; age range 35–89 years). The 93 subjects were divided into 2 groups according to whether they had a biliary dilatation or not: 17 patients did not have biliary dilatation (Group 1), whereas 76 patients had biliary dilatation (Group 2).

CT technique

All patients underwent multidetector-row CT with one of two scanners [LightSpeed Ultra (8 channel), LightSpeed VCT (64 channel); GE Healthcare, Milwaukee, WI] with the following parameters: detector collimation, 0.625–1.250 mm; table pitch, 1.0984–1.350; matrix, 512×512; and reconstruction intervals, 3.75–5.00 mm. Unenhanced scans were obtained for all patients. Contrast-enhanced scans were performed in one of three ways; one-phase, two-phase or three-phase scanning after injection of non-ionic contrast material (Iomeron 350, Bracco, Milan, Italy; Omnipaque 350, Nycomed/GE Healthcare, Cork, Ireland). The total amount of contrast material for one-phase and two-phase scanning was 100 ml administered at a rate of 3.8 ml s⁻¹, while for three-phase scanning, 120 ml was administered at a rate of 4.0 ml s⁻¹; this was followed by a 15–30 ml saline flush at a

rate of 1.5–3.0 ml s⁻¹. One-phase scanning of the portal venous phase was obtained 60 s after the injection of the contrast material. Two-phase scanning consisted of late arterial and portal venous phases. Three-phase scanning included arterial, portal venous and equilibrium phases. A bolus-tracking technique (SmartPrep; GE Healthcare) was used to determine the scan delay time for arterial and late arterial phases. For the arterial and late arterial phases, scans were initiated 15 and 18 s, respectively, after a preferred attenuation threshold (100 HU) was reached at a region of interest placed at the lumen of the abdominal aorta. Equilibrium phase images were obtained 180 s after the initiation of the contrast material injection; the reconstruction thickness and interval were both 5 mm except for the late arterial phase of two-phase scanning (3.75 mm). The numbers of patients examined with one-phase, two-phase and three-phases scanning were 43, 42 and 8, respectively.

Review of medical records

One radiologist reviewed the medical records of all 6317 patients to collect clinical and laboratory data. The presence of clinical symptoms such as abdominal pain, fever/chills and jaundice was assessed. The laboratory findings including total bilirubin level, alkaline phosphatase level, γ -glutamyltransferase level, aspartate aminotransferase (AST) level, alanine aminotransferase (ALT) level, white blood cell (WBC) count and C-reactive protein level, and these were compared between the two groups. The presence or absence of clinical symptoms on the date of hospital admission and the laboratory results on the date of CT examination were compared between the two groups. The duration of hospital stay and whether an invasive procedure was necessary were evaluated; endoscopic stone removal, endoscopic drainage, stent placement in the bile duct and percutaneous transhepatic biliary drainage were considered invasive procedures. The presence of pus, which is a diagnostic finding of acute suppurative cholangitis, was checked during endoscopic retrograde cholangiopancreatography (ERCP).

Image analysis

Axial CT scans were reviewed by consensus decision by two radiologists on a picture archiving and communication system workstation. The axial CT scans of the two groups were randomly mixed by one radiologist who did not participate in the review process.

The following image findings were assessed: (1) the presence of THADs and the extent of the THADs when present; (2) the presence of periportal tracking; (3) the presence of a periampullary duodenal diverticulum; (4) the presence of perihepatic lymphadenopathy; (5) the presence of wall thickening of the extrahepatic duct; (6) the longest transverse diameter of the gallbladder; (7) the diameter of the extrahepatic bile duct; and (8) the diameter of the largest stone in the extrahepatic bile duct.

The presence and extent of THADs were evaluated in patients for whom arterial phase scanning data were available. An area showing THADs was defined as an

Table 2. Assessment of the diameter of the extrahepatic bile duct

Extrahepatic bile duct	<50 years	51–60 years	61–70 years	>70 years	Any age with cholecystectomy
Normal (mm)	0–5.9	0–6.9	0–7.9	0–8.9	0–7.9
Borderline (mm)	6.0–7.9	7.0–8.9	8.0–9.9	9.0–10.9	8.0–9.9
Dilatation (mm)	≥8.0	≥9.0	≥10.0	≥11.0	≥10.0

area of high attenuation on the arterial phase image that returned to isoattenuation on the portal venous phase image [8]. The extent of THADs was scored by counting the number of liver segments involved (Couinaud's numbering system, segments I–VIII) [6]. The presence of periportal tracking was assessed on portal or equilibrium phase images by looking for periportal zones or halos of decreased attenuation [9]. The presence of perihepatic lymphadenopathy was determined by measuring the short-axis diameter of the nodes; a nodal diameter exceeding 7 mm at the porta hepatis, 10 mm in the portacaval space and 8 mm at the gastrohepatic ligament was regarded as abnormal [10, 11]. The diameter of the extrahepatic duct was measured perpendicular to the long axis of the extrahepatic duct. Normal or abnormal diameters of the extrahepatic duct according to age or cholecystectomy are summarised in Table 2 [12].

In patients with only dilatation of intrahepatic ducts, biliary dilatation was considered present if intrahepatic ducts were >3 mm in diameter. A thick extrahepatic bile duct wall was defined as a wall with a thickness of >1.5 mm [3]. The presence of extrahepatic duct stones or sludge was determined based on the ERCP findings. In patients with bile duct stones, the diameter of the largest stone in the extrahepatic bile duct was measured on T_2 weighted coronal MR images (2-mm slice thickness) because many patients underwent MR cholangiopancreatography (MRCP) with a 1.5-T Intera scanner (Philips Medical Systems, Best, Netherlands) at the same time (Group 1, $n=6$; Group 2, $n=22$).

Statistical analysis

Fisher's exact test, the χ^2 test and the t -test were used to compare the clinical data and imaging findings between the two groups. p -values <0.05 were considered statistically significant. Data analyses were performed using SPSS for Windows (v. 18.0; SPSS Inc., Chicago, IL).

Results

The causes of acute cholangitis in Group 1 and Group 2 are listed in Table 3 (Figures 1 and 2). The causes of acute cholangitis in Group 1 were common bile duct (CBD) stones ($n=12$) and CBD sludge ($n=3$); there were two cases with an unknown cause. Among Group 1 patients, four presented with biliary pancreatitis (CBD stones, $n=3$; CBD sludge, $n=1$). In Group 2, common causes of acute cholangitis were CBD stones ($n=41$) and biliary/peribiliary malignancies ($n=22$) (Table 3). The diagnoses of stones and sludge in the CBD were confirmed by ERCP ($n=37$), and were made based on review of CT or MR images ($n=17$). Spontaneous passage of a CBD stone was diagnosed based on ERCP

findings if the swollen duodenal papilla was lacerated without evidence of a stone in the bile duct ($n=2$). Acute pancreatitis was diagnosed clinically. Biliary and peribiliary malignancies included cholangiocarcinoma ($n=14$), pancreatic cancer ($n=5$), ampulla of Vater cancer ($n=2$) and duodenal cancer ($n=1$). Other malignancies that induced bile duct dilatation included bile duct compression by lymphadenopathy due to gastric cancer ($n=2$), bile duct dilatation due to liver metastases from a rectal cancer ($n=1$) and gallbladder cancer ($n=1$), and bile duct invasion by hepatocellular carcinoma ($n=1$). Among the 27 patients with biliary obstruction caused by tumours, 10 were diagnosed by surgery or biopsy and 17 were diagnosed by imaging findings and clinical follow-up.

Results of the comparison of the clinical and imaging findings between the two groups are summarised in Table 4. The total bilirubin levels of Group 2 were significantly higher than those of Group 1 ($p=0.001$). By contrast, Group 1 had higher median ALT levels than Group 2 ($p=0.036$). The duration of hospital stay was significantly longer in Group 2 than in Group 1 ($p<0.001$). The size of common duct stones and the diameter of the CBD were significantly larger in Group 2 than in Group 1 ($p<0.001$). Although no significant differences were found in the presence of THADs between the two groups, there was a significant difference in the extent of THADs between Group 1 and Group 2 ($p=0.003$) (Figures 3 and 4).

Discussion

Biliary dilatation accompanies acute cholangitis in the majority of acute cholangitis patients. Biliary dilatation is an important imaging finding of acute cholangitis and has been reported to be present in 73–100% of patients in previous studies [2, 3, 6]. In this study, biliary dilatation was observed in 82% of patients with acute cholangitis.

Table 3. Summary of causes of acute cholangitis in Group 1 and Group 2

Cause	Group 1 ($n=17$)	Group 2 ($n=76$)
CBD stones	8	39
CBD sludge	2	0
Passed stones	1	1
Acute pancreatitis due to CBD stones	3	1
Acute pancreatitis due to CBD sludge	1	0
Unknown	2	3
Bile duct stricture	0	3
Biliary or peribiliary malignancy	0	22
Bile duct dilatation due to other malignancy	0	5
Biliary stent malfunction in patients with cholangiocarcinoma	0	2

CBD, common bile duct.

Figure 1. A 54-year-old female with acute cholangitis (Group 1). (a) Unenhanced CT scan shows a calcified stone in the distal common bile duct (arrow). (b) Contrast-enhanced CT shows the non-dilated common hepatic duct (short arrow) and gallbladder stones (arrowhead).

However, biliary dilatation does not always accompany acute cholangitis, because biliary obstruction and acute cholangitis can exist before biliary dilatation develops [2].

The most frequent cause of acute cholangitis with or without biliary dilatation is a common duct stone [13]. Similarly, in the present study, a CBD stone was found to be the most common cause of acute cholangitis in both groups. However, all cases of acute cholangitis related to malignant conditions showed biliary dilatation. By contrast, dilatation of the bile duct was not present in any of the patients with CBD sludge ($n=3$). Four of the five patients with biliary pancreatitis belonged to Group 1, and one patient to Group 2. In terms of biliary stones, the size of CBD stones was significantly larger in Group 2 than in Group 1. The degree of biliary obstruction and dilatation might be milder in patients with smaller stones than in patients with larger stones. In addition, one study demonstrated that small stones <8 mm in size can pass through the duodenal papilla [14]; this may also

contribute to non-dilated bile ducts. In a study of the clinical significance of bile duct sludge, Keizman et al [15] reported that bile duct sludge is regarded as an early state of choledocholithiasis because 16% of patients with CBD sludge developed symptomatic CBD stones. In addition, CBD dilatation was less commonly observed in the patients with sludge in their study. On the contrary, in those patients with malignant tumours, the solid tumour masses caused a high degree of biliary obstruction, resulting in more dilatation of the biliary tree.

With regard to laboratory findings, the total bilirubin levels of the patients in Group 2 were significantly higher than those in Group 1. Hayat et al [16] reported higher median bilirubin levels in patients with malignant strictures than in those with stones. These authors argued that this may be because obstruction due to a malignant stricture tends to be more complete and more prolonged, whereas obstruction due to a stone may fluctuate according to stone movement [16]. The higher

Figure 2. A 79-year-old female with acute cholangitis (Group 2). (a) Unenhanced CT scan shows a subtle high-attenuating stone in the common bile duct (arrow). (b) Contrast-enhanced CT shows marked dilatation of the common hepatic duct (arrow).

Table 4. Results of clinical and imaging comparison between Group 1 and Group 2

Findings	Group 1 (n=17)	Group 2 (n=76)	p-value
Laboratory			
Bilirubin level (mg dl ⁻¹)	4.58 ± 2.46	8.53 ± 8.27	0.001
ALP level (IU l ⁻¹)	316.47 ± 474.94	322.77 ± 258.88	0.939
GGT level (IU l ⁻¹)	569.06 ± 315.44	570.42 ± 646.05	0.994
AST level (IU l ⁻¹)	245.35 ± 184.03	175.68 ± 155.78	0.110
ALT level (IU l ⁻¹)	256.71 ± 179.90	170.19 ± 145.07	0.036
WBC count (cells µl ⁻¹)	11596.35 ± 3052.99	14245.45 ± 9762.96	0.052
CRP concentration (mg l ⁻¹)	101.91 ± 81.92	106.48 ± 151.70	0.912
Non-laboratory			
Age (years)	67.12 ± 12.92	69.38 ± 10.38	0.439
Sex (M:F)	11:6	48:28	0.905
Abdominal pain	16	63	0.449
Fever	9	43	0.785
Jaundice	16	67	>0.100
Invasive treatment	14	67	0.455
Pus on ERCP	2 (14)	3 (60)	0.237
Admission days	8.12 ± 2.31	16.99 ± 13.75	<0.001
CT findings			
Presence of THAD	9 (9)	36 (41)	0.570
Extent of THAD	4.49 ± 2.59	7.69 ± 1.06	0.003
Lymphadenopathy	6	33	0.512
Periportal tracking	6	19	0.757
Periampullary diverticulum	7	18	0.125
Wall thickening of common bile duct	3 (17)	14 (60)	0.749
Bile duct diameter (mm)	7.71 ± 1.10 (17)	16.05 ± 4.91 (63)	<0.001
Longest transverse diameter of gallbladder (mm)	35.44 ± 13.95 (16)	38.74 ± 11.80 (58)	0.344
Stone size (mm)	5.58 ± 1.11 (6)	12.63 ± 4.52 (22)	<0.001

ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; ERCP, endoscopic retrograde cholangiopancreatography; F, female; GGT, γ -glutamyltransferase; M, male; THAD, transient hepatic attenuation differences; WBC white blood cell.

Values are given as mean $\pm$ standard deviation or number unless otherwise indicated.

Values in parentheses are available numbers.

p-values <0.05 were considered statistically significant.

bilirubin levels observed in Group 2 in our study compared with Group 1 was probably caused by malignant obstruction and larger stones in the former group, which could lead to more prolonged and complete obstruction of the bile ducts.

Group 1 had significant higher median ALT levels than Group 2 in this study. In addition, the median AST

level was higher in Group 1 than in Group 2, although this difference was not statistically significant. In patients with biliary obstruction, elevation of serum transaminase activity (AST/ALT levels), which is a sensitive indicator of hepatocellular damage, is probably due to liver cell injury caused by bile duct obstruction [16, 17]. Several studies have shown that marked elevation of serum

Figure 3. A 76-year-old-male with acute cholangitis caused by biliary pancreatitis (Group 1). (a, b) Arterial phase CT scans show focal enhancement in the liver parenchyma around the gallbladder fossa (arrows). Two segments showed transient hepatic attenuation difference involvement. Also note peripancreatic fluid collection (arrowheads).

Figure 4. A 78-year-old male with acute cholangitis caused by a common bile duct stone (Group 2). (a, b) Arterial phase CT scans show diffuse mottled enhancement of the entire liver parenchyma and dilatation of the intrahepatic bile duct (arrows on b). Eight segments showed transient hepatic attenuation difference involvement.

transaminase levels is related to biliary pancreatitis, passage of stones through the duodenal papilla, acute biliary stone disease and malignant biliary strictures [16–19]. In a previous study of patients with CBD stones showing high levels of AST/ALT, these serological features were frequently observed in the patients with a smaller diameter of CBD [20]. These investigators thought that the degree of elevation of intraductal pressure might be higher in younger patients with a small diameter of CBD than in older patients in whom senile dilatation of CBD was usually observed. In this study, however, the mean ages were not significantly different between the two groups. Although the pathological mechanism for marked elevation of AST/ALT levels in Group 1 was not known exactly, we suppose that it can be explained by the difference in the obstructing causes between the two groups, *i.e.* biliary obstruction induced by malignant tumours or large stones in Group 2 might lead to more prolonged dilatation of the bile duct, and this might result in increased flexibility in the bile duct with physiological adaptation to the raised intraductal pressure.

Factors relevant to adverse clinical outcomes in patients with acute cholangitis are high bilirubin levels, high WBC counts, bacteraemia and a malignant cause of biliary obstruction [21, 22]. Patients with malignant obstruction have a higher risk of death due to complete biliary obstruction leading to elevated ductal pressures with increased cholangiovenous reflux [23]. In this study, the length of hospital stay was significantly longer for Group 2 patients than for Group 1 patients. Moreover, all patients with malignant biliary obstruction were classified into Group 2, and there was a tendency for the WBC count to be higher in Group 2 patients than in those in Group 1, although this difference was not statistically significant ($p=0.052$). In addition, the total bilirubin levels of Group 2 patients were significantly higher than in those in Group 1. These findings are consistent with those of previous studies.

Several studies have described the imaging findings of acute cholangitis [2–6]. In studies based on CT or MRI,

bile duct dilatation, wall thickening of the bile duct, the presence of biliary obstructive lesions and areas of hyperintense signal on T_2 weighted images or THADs on dynamic-enhanced CT images were observed in patients with acute cholangitis [2–6]. In one study that used CT to discriminate between suppurative cholangitis and non-suppurative cholangitis, papillitis and THADs were found to be the most significant predictors of acute suppurative cholangitis [5]. In our study, biliary dilatation and THADs were observed in 82% and 90% of patients, respectively. However, the extent of THADs was the only significant discriminative CT finding between acute cholangitis patients with and without biliary dilatation.

THADs arise as a result of an increase in arterial blood supply, usually as a compensatory reaction to a decrease in portal flow [4, 6, 24–26]. The portal vein and hepatic artery communicate mainly through the sinusoid system and peribiliary plexus; these shunts attempt to compensate locally for reduced portal inflow and the arterial reaction is proportional to the portal lessening [24]. THADs are produced frequently in the setting of bile duct dilatation by obstructive lesions [6, 24, 26]. Dilatation of the bile ducts causes collapse of the peribiliary plexus owing to the lack of a muscular wall [24]. The collapsed peribiliary plexus induces a decrease in blood flow from the portal vein into the sinusoid, which results in arterial compensation because of the partial depletion of portal inflow [4, 6, 24–26]. In a study of THADs in patients with biliary dilatation, the extent of THADs was greater in the cholangitis group than in the non-cholangitis group [6]. By contrast, in the current study of patients with acute cholangitis, the extent of THADs was greater in the group with biliary dilatation than in the group without biliary dilatation, although the incidence of THADs was not different between these two groups. Collapse of the peribiliary plexus by biliary dilatation might be an important basic mechanism for the different extents of THADs between the two groups. These results as well as those reported previously [6] suggest that biliary dilatation and biliary infection are

two factors that could determine the extent of THADs in patients with acute cholangitis.

There are several limitations to our study. First, some cases of biliary obstruction were diagnosed by imaging findings and clinical follow-up. However, if we had included only patients diagnosed by surgery or biopsy, there is the possibility that patients with acute cholangitis without biliary dilatation would not have been included, which might have resulted in a different kind of selection bias. Second, we only included patients who underwent enhanced CT scans. There is the possibility that patients with a mild degree of acute cholangitis who did not undergo further imaging studies or treatment were not included in the study. In addition, exclusion of patients diagnosed by ultrasonography or MRCP may have affected our results.

In conclusion, the most common causes of acute cholangitis without biliary dilatation were CBD stones and sludge. Some of these patients also presented with biliary pancreatitis. Patients with acute cholangitis without biliary dilatation had a lower level of bilirubin, a higher level of ALT, and a shorter duration of hospital stay than patients with acute cholangitis with biliary dilatation. The extent of THADs was the only CT finding that discriminated between the two groups.

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