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Journal

Journal of Ultrasound in Medicine, 42(6)

ISSN

0278-4297

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Publication Date

2023-06-01

DOI

10.1002/jum.16138

Peer reviewed



Published in final edited form as:

J Ultrasound Med. 2023 June ; 42(6): 1257–1265. doi:10.1002/jum.16138.

Sonographic assessment of acute versus chronic cholecystitis: an ultrasound probability stratification model

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Abstract

Objectives: What sonographic variables are most predictive for acute cholecystitis? What variables differentiate acute and chronic cholecystitis?

Methods: The surgical pathology database was reviewed to identify adult patients who underwent cholecystectomy for cholecystitis and had a preceding ultrasound of the right upper quadrant within 7 days. 236 patients were included in the study. A comprehensive imaging review was performed to assess for gallstones, gallbladder wall thickening, gallbladder distension, pericholecystic fluid, gallstone mobility, the sonographic Murphy's sign, mural hyperemia, and the common hepatic artery peak systolic velocity.

Results: Of 236 patients with a cholecystectomy, 119 had acute cholecystitis, and 117 had chronic cholecystitis on surgical pathology. Statistical models were created for prediction. The simple model consists of three sonographic variables and has a sensitivity of 60% and specificity of 83% in predicting acute versus chronic cholecystitis. The most predictive variables for acute cholecystitis were elevated common hepatic artery peak systolic velocity, gallbladder distension and gallbladder mural abnormalities. If a patient had all three of these findings on their preoperative ultrasound, the patient had a 96% chance of having acute cholecystitis. Two of these variables gave a 73-93% chance of having acute cholecystitis. One of the three variables gave a 40-76% chance of having acute cholecystitis. If the patient had 0 of 3 of the predictor variables, there was a 29% chance of having acute cholecystitis.

Conclusions: Gallbladder distension, gallbladder mural abnormalities and elevated common hepatic artery peak systolic velocity are the most important sonographic variables in predicting acute versus chronic cholecystitis.

Introduction

Acute cholecystitis is typically caused by obstructing gallstones and is a common reason for hospitalization. More than 500,000 cholecystectomies are performed annually in the United

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States.¹ The management of acute cholecystitis is surgical, generally with early laparoscopic cholecystectomy.^{2,3}

Imaging plays a critical role in the diagnosis of acute cholecystitis, with ultrasound serving as the first-line modality.⁴ Ultrasound has a wide range of reported sensitivity and specificity in the diagnosis of acute cholecystitis, which may be secondary to varying institutional image quality, improved ultrasound resolution over time, and variability in what sonographic factors are considered “positive” for acute cholecystitis.⁵⁻⁷ Many such variables have been assessed, including the presence of gallstones, gallbladder wall thickening, gallbladder distension, pericholecystic fluid, gallstone mobility, the presence of the sonographic Murphy’s sign, hyperemia involving the gallbladder wall, and the elevation of the peak systolic velocity of the common hepatic artery velocity. While all these factors contribute to the diagnosis of cholecystitis, the utility of these variables has generally focused on distinguishing cholecystitis versus no cholecystitis, rather than acute versus chronic cholecystitis.

Therefore, we sought to investigate which sonographic variables most contributed to the diagnosis of acute versus chronic cholecystitis.

Materials and methods

Patient population

This study was approved by our institutional review board with the requirement for informed patient consent waived owing to the retrospective nature of the study. We reviewed the surgical pathology database from 2017 to 2019 to identify adult patients (aged 18 and over) who underwent cholecystectomy for acute or chronic cholecystitis and who had a preceding ultrasound examination of the right upper quadrant within the 7 days prior to surgery. A total of 236 patients were included in the study (mean age 46.2 years; 70 male, 166 female). In order to ensure the concordance of the ultrasound findings with the pathology findings, we limited the time between ultrasound and surgery to be 7 days based on pathologic studies and surgical guidelines.^{8,9} The average length of time between ultrasound and surgery was 1.9 days, standard deviation 2.18 days, range 0-7 days.

Sample size justification: A sample of 236 patients (119 acute cholecystectomy and 117 chronic cholecystectomy) has 91% power to detect an increase of 0.1 from the area under the receiver operating characteristic curve (AUC) under the null hypothesis of 0.65 to an AUC of 0.75 (under alternative hypothesis) using a one-sided z-test at a significance level of 0.05.

Ultrasound technique

Each ultrasound was performed by an American Registry for Diagnostic Medical Sonography certified sonographer. GE Logiq E9 ultrasound machines (GE Healthcare, Waukesha, WI) were used for the exam. Greyscale and color Doppler views of the gallbladder were obtained using either the 9L or C5-1 probes. All ultrasounds were formally performed by the sonographer. No POCUS exams were included. Patients were scanned without deference to post-prandial state. The gallbladder was assessed with the patient in

the supine position in the longitudinal and transverse planes, and in the left lateral decubitus positioning if gallstones were present. The gallbladder wall was measured in the transverse plane, with the transducer parallel to the gallbladder wall.

The hepatic artery peak systolic velocity was measured by angle corrected spectral Doppler with the angle of insonation less than or equal to 60 degrees, where it runs parallel to the portal vein.

Imaging review

Imaging variables included cholelithiasis, gallbladder wall thickness, hepatic artery peak systolic velocity, gallbladder wall abnormalities, mural hyperemia, pericholecystic fluid, gallbladder distension, gallstones in the neck, immobile stones, and the sonographic Murphy's sign. Imaging variables were assessed by retrospective review of an abdominal fellowship-trained radiologist with 2 years of post-fellowship experience who was blinded to the pathology results.

Measurement of the proper hepatic artery peak systolic velocity was considered adequate when the vessel was measured parallel to the main portal vein as it courses in the hepatoduodenal ligament¹⁰. If the hepatic artery peak systolic velocity was measured inaccurately, those data points were not included; 37 data points were excluded from the analysis. The sonographic Murphy's sign, or the point of maximal tenderness elicited when the ultrasound probe is held over the gallbladder was reported as positive or negative by the sonographer.^{6,11-13} Gallbladder wall thickening was defined as greater than 3 mm, measured in the transverse plane with the transducer parallel to the gallbladder wall.^{6,14-16} Abnormal gallbladder distension was defined as equal to or greater than 4 cm in short axis dimension by 10 cm in long axis dimension.^{13,17,18} Gallbladder wall abnormalities were defined as any irregularity to the wall, including discontinuity or focal thickening.¹⁵ Gallbladder mural hyperemia was assessed on color or power Doppler, and any flow in the gallbladder wall was considered positive.^{14,16,19,20}

Statistical analysis

Patient characteristics and ultrasound parameters were reported using counts (proportions) for categorical variables and mean (standard deviation) for continuous variables. Univariate analysis was performed to compare variables between the two groups (acute versus chronic cholecystitis), using Wilcoxon rank-sum tests for continuous variables or χ^2 tests (or Fisher exact tests when count ≤ 5) for categorical variables.

Model 1: the full model—Multivariable logistic regressions were used to estimate the probability of acute versus chronic cholecystitis, which included all investigated ultrasound parameters as predictors. Sensitivity and specificity of the predicted classification were computed based on the fitted model, with the optimal cut-off value chosen based on Youden index.²¹

Model 2: the reduced-variable model—We created a second model by identifying predictor variables that could be eliminated from the full models in a backward selection

while maintaining discriminatory accuracy in prediction. The discriminatory accuracy of prediction was summarized using area under the receiver operating characteristic curve (AUC) using all data. We successively dropped one variable with the smallest reduction (or largest gain) of AUC, ending when dropping any variable from the reduced model would lead to AUCs at least 0.03 lower than the AUC of full model. Note that, the variable selection procedure focused on differentiating acute versus chronic cholecystitis, and hence is based on whether retaining a predictor is able to improve discriminatory accuracy (i.e., AUC) instead of the statistical significance of this predictor. Although hepatic artery velocity is not statistically significant, AUC was improved by retaining this predictor. On the other hand, some predictors found statistically significant in univariate and multivariate analysis did not improve AUC noticeably and thus were removed.

Model 3: the simple model—We probed the continuous predictor variables that had remained in model 2 for optimal cut-off values, by assessing AUC for each continuous variable at different cut-off values. The cut-off value with the highest AUC was chosen in the final simple model. This third model was created by using the categorized predictor variables according to the identified cut-off values to develop a simple classification rule to predict acute cholecystitis versus chronic cholecystitis.

We compared the AUC from the models fit using the full data to AUCs from the models fit using 5-fold cross-validation, and the differences between them (optimism) were used to evaluate overfitting of the models (severe overfitting would lead to large optimism). The optimisms for full, reduced variable, and simple models were 0.048, 0.001, and 0.003, respectively, which indicated that the full model was overfitted by including too many unnecessary predictors, while the reduced variable and simple models did not suffer from overfitting. The cross-validation AUC is adjusted for overfitting.

Statistical analysis was performed using SAS 9.4 (SAS Institute Inc., Cary, NC) and R version 4.0.4 (R Foundation for Statistical Computing, Vienna, Austria). A two-sided $P < 0.05$ was used to determine statistical significance.

Results

Of our patient population of 236 patients with a cholecystectomy, 119 had acute cholecystitis, and 117 had chronic cholecystitis based on final surgical pathology. Patients with gangrenous cholecystitis by pathology were included in the “acute” category.²²

In univariate analysis, the following investigated variables were significant in differentiating acute from chronic cholecystitis: gallbladder wall thickness, mural hyperemia, gallbladder mural abnormalities, gallbladder distension, pericholecystic fluid and the sonographic Murphy’s sign. Patients’ age and gender were also significantly different. Cholelithiasis, GS in neck, gallstone immobility, and hepatic artery velocity were not significantly different between acute and chronic cholecystitis. (Table 1)

The full model

The full model includes all variables and has a sensitivity of 70% and a specificity of 80% for differentiating between acute and chronic cholecystitis. The full model's AUC using all data was 0.768 and AUC by cross-validation was 0.720 (adjusted for overfitting), which suggests inclusion of too many unnecessary predictors (Supplementary Table 1, Figure 1).

The reduced variable model

The reduced variable model demonstrated a similar accuracy to the full model while dropping less predictive variables. Final model included hepatic artery peak systolic velocity (HAV), gallbladder distension, and mural abnormalities (details in Supplementary Table 2). Although the association between the hepatic artery peak systolic velocity and acute cholecystitis was not significant, including hepatic artery peak systolic velocity improved AUC and hence it was retained in the model. The reduced variable model's AUC using all data was 0.751 and adjusted AUC by cross-validation was 0.750 (Supplementary Table 1, Figure 1). The reduced variate model of $0.0043 \times \text{HAV (in cm/sec)} + 1.416 \times \text{gallbladder distension} + 2.109 \times \text{mural abnormalities} - 1.695$ (cut-off point selected by Youden Index) was able to predict acute cholecystitis (from chronic cholecystitis) with sensitivity of 59% and specificity of 85%.

The simple model

The simple model further dichotomized the continuous variable (hepatic artery peak systolic velocity with cut point of greater than 96 cm/second) in the reduced model, leading to a more clinically practical model while preserving the discriminatory accuracy. The simple model's AUC using all data was 0.753 and adjusted AUC by cross-validation was 0.750 (Supplementary Table 1, Figure 1). The simple model of $0.497 \times (\text{HAV} > 96 \text{ cm/sec}) + 1.410 \times \text{gallbladder distension} + 2.099 \times \text{mural abnormalities}$ (cut-off point selected by Youden Index) was able to predict acute cholecystitis (from chronic cholecystitis) with sensitivity of 60% and specificity of 83%. The most discriminative variables for acute or chronic cholecystitis were hepatic artery peak systolic velocity >96 cm/second, presence of gallbladder distension, and gallbladder mural abnormalities. If these three variables were present, the patient has a 96% chance of having acute cholecystitis. Different combinations yielded 8 stratification categories. If the patient had 0/3 of these variables, there was a 29% risk of having acute cholecystitis. If the patient had 1/3 of these variables, they were at intermediate risk (40-76%) for having acute cholecystitis. If the patient had 2/3 of these variables, they were at high risk (73-93%) for having acute cholecystitis (Table 2).

A consideration to each of the three sonographic variable components included in the simple model is worthwhile. Although the association between the hepatic artery peak systolic velocity and acute cholecystitis was not significant on univariate analysis, hepatic artery peak systolic velocity >96 cm/second increased the simple model's accuracy. A patient with gallbladder distension has 4 times the odds to have acute cholecystitis (odds ratio: 4.09, 95% CI: 1.85, 9.07). A patient with gallbladder wall abnormalities has eight times the odds to have acute cholecystitis (odds ratio: 8.16, 95% CI: 3.34, 19.93) (Supplementary Table 3). The simple model consists of two objective values (gallbladder distension, hepatic artery peak systolic velocity) and one subjective value (gallbladder wall abnormalities).

Discussion

We found that three sonographic variables most contributed to the sonographic distinction of acute versus chronic cholecystitis: hepatic artery velocity greater than or equal to 96 cm/second, gallbladder distension, and gallbladder mural abnormalities. If all three variables are identified, the patient has a 95% chance of having acute cholecystitis. This reduced variable model demonstrates a fair performance with a sensitivity and specificity in line with the literature's overall assessment of ultrasound as a modality for diagnosis of acute cholecystitis.^{23,24} Further, by only including three imaging variables, strength of the simple model is a facile tool for rapid image interpretation in the acute setting with each of the three variables rapidly increasing the overall likelihood of acute cholecystitis.

Gallbladder mural abnormalities was the strongest differentiating variable between acute and chronic cholecystitis in our study, with an odds ratio of 8.24. Given that gallbladder wall thickening is nonspecific and can be seen in other etiologies such as hypoalbuminemia or hepatitis, the search for mural discontinuity or irregular mural thickening can help in the diagnosis of acute cholecystitis (figure 2).^{15,17}

Gallbladder distension, which is presumably caused by increasing intraluminal pressure after cystic duct obstruction by a gallbladder stone, was an additional important variable in the differentiation between acute and chronic cholecystitis, with an odds ratio of 4.12 (figure 3a and 3b). This reflects other studies that have shown that distension of the gallbladder is associated with acute cholecystitis.^{13,17,18,25} Suggested cut off levels for distension are variable, and the literature often references 8 cm longitudinal by 4 cm transverse, while others argue for a subjective characterization of gallbladder distension given the variability in gallbladder shape.²⁶ There are rare other causes of gallbladder distension, including gallbladder torsion or volvulus, making acute cholecystitis a much more common and important diagnostic consideration.²⁷

Hepatic artery peak systolic velocity has been shown to be a predictor of acute cholecystitis (figure 4).^{10,28} We have been routinely measuring hepatic artery velocity at our institution for right upper quadrant exams since 2018 and have found it a useful diagnostic tool. Interestingly, although the elevated hepatic artery velocity was not statistically significant as a discriminatory variable, including it in the model yielded improved accuracy. The retrospective review of the images included strict hepatic artery velocity quality control, with values not meeting the standard being excluded from the analysis. In total, 37 of these data points were excluded, both identifying a quality improvement project for our sonographers and suggesting a reason that the HAV improved the predictive ability of the model but also was not statistically significant.

Elevated hepatic artery velocity above 100 cm/second can help distinguish between structural (i.e. cholangitis and cholecystitis) and non-structural causes (i.e. drug induced hepatitis) of elevations in liver function tests.^{28,29} Elevated hepatic artery velocity is not specific for acute cholecystitis and can be elevated in other causes of hepatic hyperemia including infectious or inflammatory conditions. A markedly elevated hepatic artery velocity greater than 200 cm/second is not necessarily indicative of primary hepatobiliary disease but

may reflect other hepatic injury related to systemic causes such as sepsis.³⁰ Recent research on elevation of the cystic artery velocity may be more predictive of acute cholecystitis as it is less affected by other pathologic or physiologic states than the hepatic artery velocity.³¹

The significance in predicting acute versus chronic cholecystitis comes down to better diagnostic information for the surgeons allowing for additional support if needed, and better inform the consent process if there is prediction of conversion to open. Acute cholecystitis patients are more likely to be converted from laparoscopic to open surgery compared to those which chronic cholecystitis.³² Chronic cholecystitis, however, occurs after repeated episodes of inflammation and yields fibrosis on histologic analysis and can portend increased technical difficulty in surgery.³³⁻³⁵ conversion from laparoscopic to open cholecystectomy is associated with longer hospital stays and increased morbidity.^{36,37} Further, differentiating between acute versus chronic may allow delayed or outpatient surgery rather than emergent/urgent inpatient surgery, which may thereby decrease the costs of medical care for this very common entity.^{38,39}

We acknowledge limitations to our study. Our use of surgical pathology as the gold standard does limit the generalizability of our study as nearly all of our patient population had an acute presentation, were symptomatic, and found on pathology to have some inflammation of the gallbladder. We did not include clinical variables such as fever or leukocytosis, instead preferring to focus the paper on sonographic variables. Further, although efforts were made to adhere to strict imaging criteria for each sonographic finding, this is a single reader study.

Conclusion

Elevated peak systolic hepatic artery velocity, gallbladder distension, and gallbladder wall abnormalities are highly predictive for acute versus chronic cholecystitis. This simple model may improve rapid characterization of imaging findings and allow for improved diagnostic confidence and timely communication with the surgical team.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgement

The project was partly supported by the National Center for Advancing Translational Sciences, National Institutes of Health through grant # UL1 TR001860.

The authors acknowledge Julie Ostoich-Prather for her assistance in figure preparation for this manuscript.

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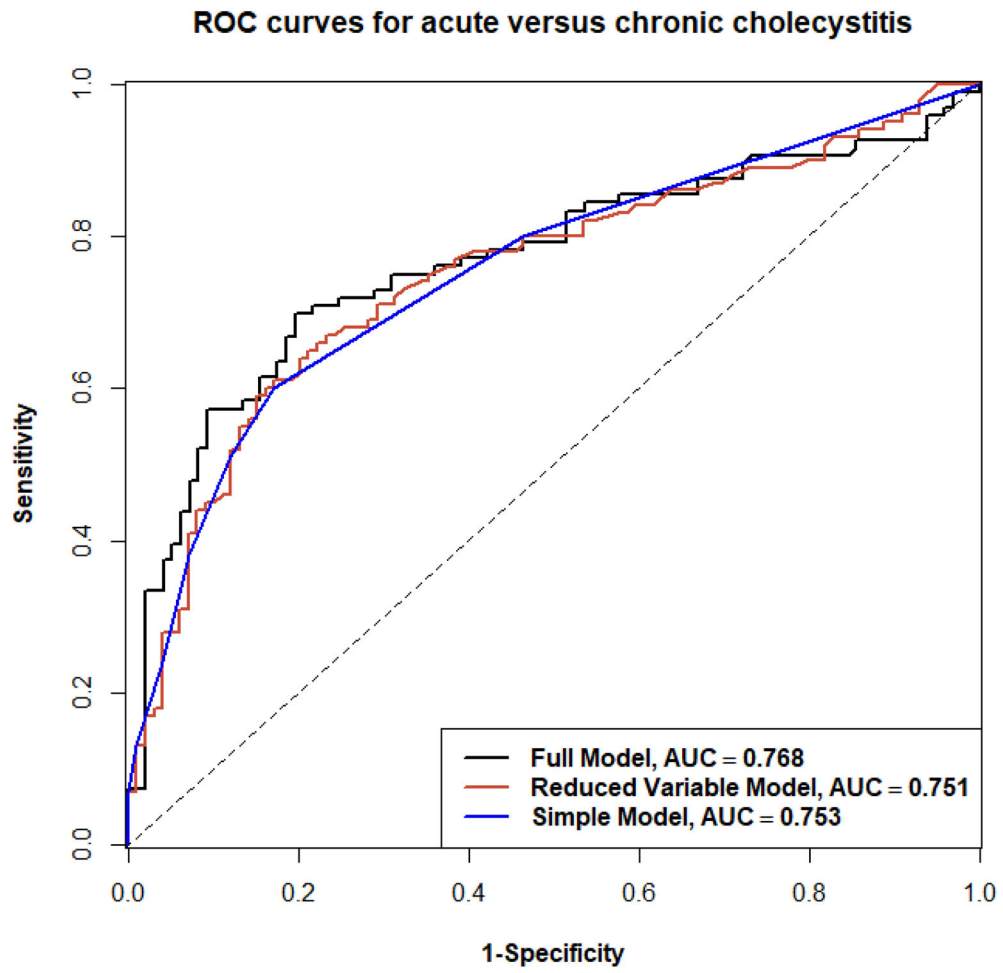


Figure 1. ROC curves of different models for acute versus chronic cholecystitis using all patients.



Figure 2: Transabdominal sagittal ultrasound of the gallbladder showing marked wall thickening (calipers). This gallbladder wall measures at 12 mm and normal is less than 3mm. In addition, this gallbladder demonstrates mural abnormalities (arrow), demonstrated by an irregular, striated appearance of the gallbladder wall.

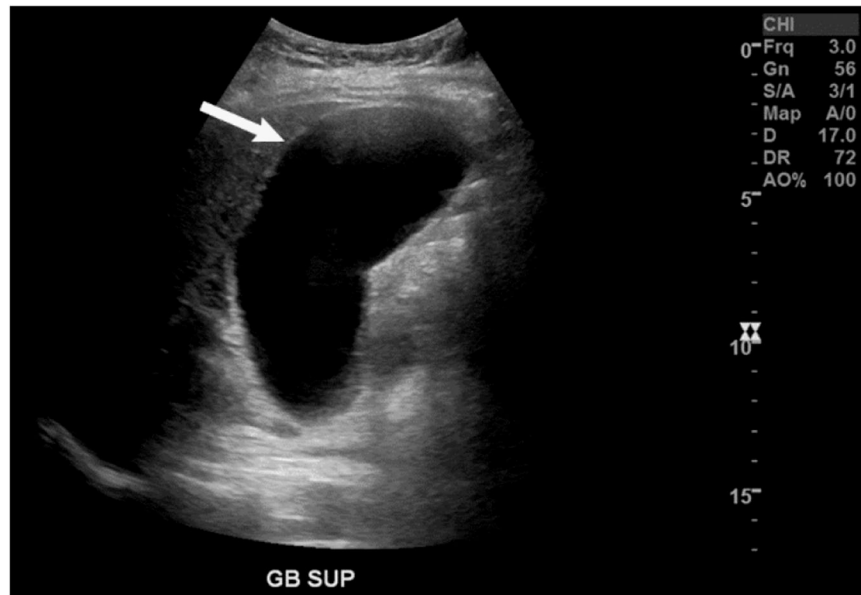


Figure 3a: Transabdominal sagittal ultrasound of the gallbladder (arrow) shows marked luminal distension, with long axis measurement of 10 cm.

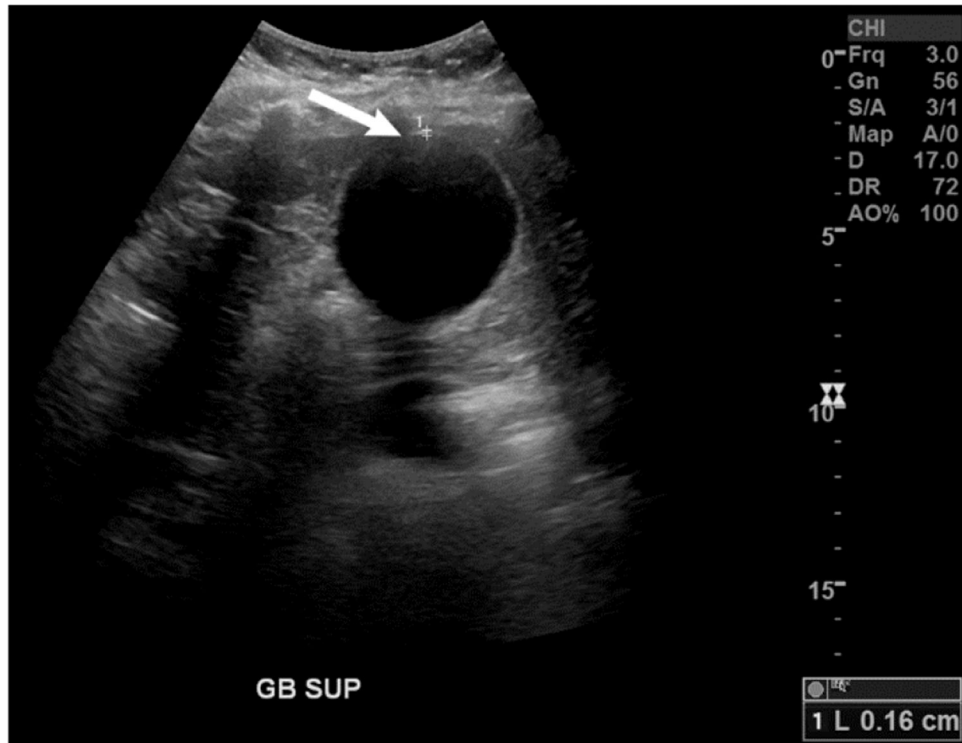


Figure 3b: Transabdominal transverse ultrasound of the gallbladder shows a thin gallbladder wall (arrow), but with luminal distension to 5.1 cm measured on the short axis.

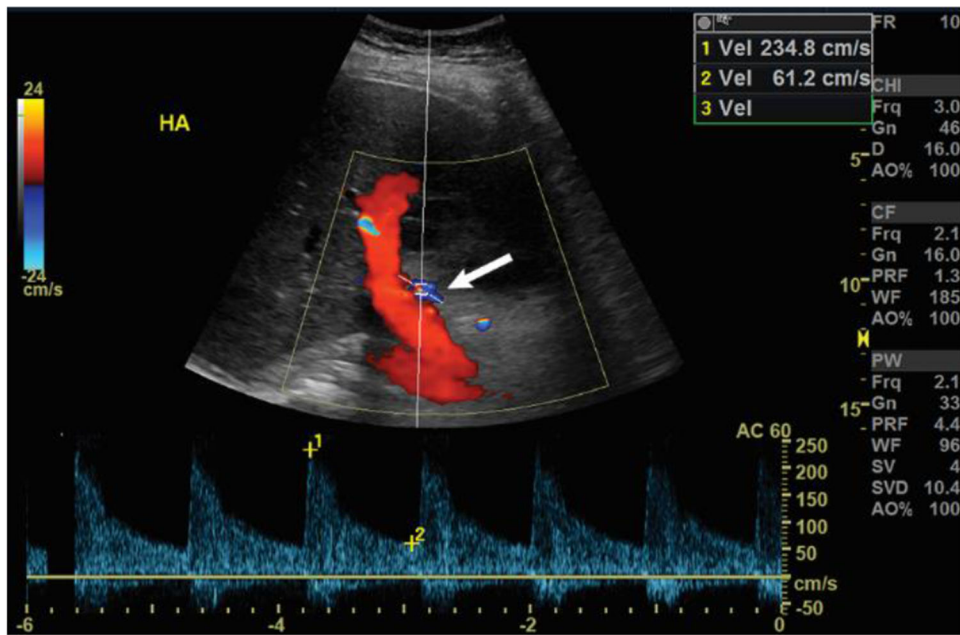


Figure 4: Transabdominal sagittal spectral Doppler ultrasound image at the porta hepatis showing the common hepatic artery being appropriately measured, with angle correction and measured parallel to the portal vein (red vessel). This hepatic velocity artery is elevated at 234.8 cm/second.

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Table 1.

Univariate analysis of all variables

	All patients (n=236)	Acute (n=119)	Chronic (n=117)	
Variable	Mean (\pm SD) or n (%)	Mean (\pm SD) or n (%)	Mean (\pm SD) or n (%)	P-value
GB wall thickness (in mm, n=235)	3.3 \pm 1.9	3.9 \pm 2.2	2.8 \pm 1.4	<0.001
HAV (in cm/sec, n=199)	98.9 \pm 46.6	103.9 \pm 49.6	93.8 \pm 42.9	0.109
Age (in year, n=236)	46.2 \pm 17.3	49.3 \pm 17.0	43.1 \pm 17.0	0.004
Cholelithiasis				0.568
Yes	224 (94.9)	114 (95.8)	110 (94.0)	
No	12 (5.1)	5 (4.2)	7 (6.0)	
GS in neck				0.641
Yes	162 (68.9)	83 (70.3)	79 (67.5)	
No	73 (31.1)	35 (29.7)	38 (32.5)	
Immobile GS				0.708
Yes	100 (42.4)	49 (41.2)	51 (43.6)	
No	136 (57.6)	70 (58.8)	66 (56.4)	
Murphy's sign				0.022
Yes	92 (39.0)	55 (46.2)	37 (31.6)	
No	144 (61.0)	64 (53.8)	80 (68.4)	
Peri-GB fluid				0.001
Yes	36 (15.3)	27 (22.9)	9 (7.7)	
No	199 (84.7)	91 (77.1)	108 (92.3)	
GB distension				<0.001
Yes	55 (23.3)	41 (34.5)	14 (12.0)	
No	181 (76.7)	78 (65.6)	103 (88.0)	
Wall abnormality				<0.001
Yes	54 (22.9)	45 (37.8)	9 (7.7)	
No	182 (77.1)	74 (62.2)	108 (92.3)	
Mural hyperemia				0.005
Yes	26 (11.2)	20 (17.0)	6 (5.2)	
No	207 (88.8)	98 (83.1)	109 (94.8)	
Gender				<0.001
Male	70 (29.7)	48 (40.3)	22 (18.8)	
Female	166 (70.3)	71 (59.7)	95 (81.2)	

Note: Variables were compared between acute versus chronic cholecystitis using Wilcoxon rank-sum tests for continuous variables or χ^2 tests (or fisher exact tests when count ≤ 5) for categorical variables.

Table 2.

Estimated probability of acute cholecystitis (versus chronic cholecystitis) based on the simple model (n = 199)

Ultrasound Parameters Included in the Simple Model			Probability of Acute Cholecystitis (%)
HAV (in cm/sec)	Gallbladder Distension	Wall Abnormalities	
<96	No	No	29
		Yes	76
	Yes	No	62
		Yes	93
96	No	No	40
		Yes	84
	Yes	No	73
		Yes	96

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