Published in final edited form as:

J Gastrointest Surg. 2014 October ; 18(10): 1777–1783. doi:10.1007/s11605-014-2599-9.

Predictors of Negative Intraoperative Findings at Emergent Laparotomy in Patients with Cirrhosis

Elliot B. Tapper,

Department of Medicine, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Boston, MA 02215, USA. Division of Gastroenterology and Hepatology, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Boston, MA 02215, USA

Vilas Patwardhan,

Division of Gastroenterology and Hepatology, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Boston, MA 02215, USA

Laura M. Mazer,

Department of Surgery, Beth Israel Deaconess Medical Center, Boston, MA, USA

Byron Vaughn,

Division of Gastroenterology and Hepatology, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Boston, MA 02215, USA

Gail Piatkowski,

Decision Support, Beth Israel Deaconess Medical Center, Boston, MA, USA

Amy R. Evenson, and

Department of Surgery, Beth Israel Deaconess Medical Center, Boston, MA, USA

Raza Malik

Division of Gastroenterology and Hepatology, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Boston, MA 02215, USA

Abstract

Background—Emergent surgery in the setting of decompensated cirrhosis is highly morbid. We sought to determine the clinical factors associated with negative intraoperative findings at emergent laparotomy.

Methods—We performed a retrospective cohort study of consecutive inpatients with a diagnosis of cirrhosis (ICD-9 571) admitted to the Beth Israel Deaconess Medical Center (Boston, MA) who underwent emergent, nonhepatic, abdominal surgery between May 6, 2005 and September 3, 2012.

Results—Eighty-six patients with cirrhosis were included with a mean model for end-stage liver disease score of 21.3 ± 7.95 and a 90-day mortality rate of 39.5 %. Twelve (16.2 %) patients had negative laparotomies. Negative intraoperative findings were independently associated with (1)

Correspondence to: Elliot B. Tapper.

Electronic supplementary material The online version of this article (doi:10.1007/s11605-014-2599-9) contains supplementary material, which is available to authorized users.

paracentesis prior to a preoperative diagnosis of perforated viscus ($P=0.006$), (2) development of an indication for emergent surgery after 24 h into hospital admission for another reason ($P=0.020$), and (3) a preoperative diagnosis of bowel ischemia ($P=0.005$), with odds ratios of 10.1 (CI 1.92–66.83), 5.80 (CI 1.32–33.39), and 11.1 (CI 2.08–77.4), respectively. Free air on computed tomography (CT) imaging was found in 64.3 % (9/14) of patients who had a paracentesis within the preceding 48 h compared to 10.1 % (7/72) among patients who did not undergo a paracentesis ($P<0.001$). Only 45 % of patients with free air following a paracentesis had positive findings at laparotomy compared to 100 % in those without a preceding paracentesis ($P=0.038$). Negative laparotomy was independently predictive of in-hospital mortality (OR 4.7; $P=0.034$).

Conclusion—The possibility of a negative laparotomy is suggested by preoperative clinical factors. In particular, free air following a paracentesis does not necessarily indicate that operative intervention is required. Consideration of close observation before laparotomy in these patients is reasonable.

Keywords

Liver disease; Paracentesis; Acute-on-chronic liver failure; Bowel ischemia

Introduction

The morbidity and mortality of surgery in patients with cirrhosis can be striking. With a relatively high risk of mortality following even minor procedures, there is no such thing as a benign operation in the setting of cirrhosis.^{1–5} Not surprisingly, when occult advanced liver disease is discovered at the time of laparotomy, the planned procedure is often aborted. However, high perioperative mortality remains for even exploratory laparotomy alone.^{6–8}

Accordingly, much attention has been paid toward stratifying cirrhotic patients by perioperative risk. The Model for End-Stage Liver Disease (MELD) score, American Society of Anesthesiologists (ASA) class, and albumin level are clinical predictors that have best forecast postoperative mortality.^{9–12} Yet, even for those patients with adverse predictors, a presentation of profound abdominal pain, leukocytosis, lactic acidosis, or imaging findings suggestive of impending abdominal catastrophe may compel the surgeon, particularly in situations where an operation appears the difference between life and death.

The risk of death for cirrhotic patients after emergency surgery can be as high as 80 %.^{13,14} While the literature has described outcomes after emergent surgery,^{7,9,10} little attention is paid to the role of preceding clinical events on intraoperative findings. Some emergent surgery, namely those that are so-called ‘negative laparotomies’, may be preventable. Herein, we present a review of our experience with emergent laparotomy in cirrhotic patients in order to identify predictors of potentially avoidable negative laparotomies.

Methods

Study Design and Data Collection

We performed a retrospective cohort study of consecutive inpatients with a diagnosis of cirrhosis (ICD-9 571) admitted to the Beth Israel Deaconess Medical Center (Boston, MA)

who underwent emergent, nonhepatic, abdominal surgery between May 6, 2005 and September 3, 2012. Clinical and demographic data were obtained from the electronic medical record. Cirrhosis was confirmed by biopsy pathology where available, clinical factors including radiographic liver morphology and evidence of portal hypertension (ascites, varices, splenomegaly), and expert consensus as documented by both medical hepatology and surgical services. A surgery was determined to be emergent based on review of the intraoperative anesthesiology record. The following demographic and clinical data were obtained by chart review: (1) laboratory test results, (2) ASA class, (3) type of surgery and operative indication, (4) preoperative computed tomography (CT) reports, (5) nonoperative preoperative interventional procedures (e.g., paracentesis), (6) intensive care unit (ICU) admission and length of ICU stay, (7) overall length of stay, (8) 30-day readmission, and (9) patient disposition including in-hospital mortality. Follow-up was censored at the date of last known contact, liver transplantation, or death. CT scans were analyzed if obtained within 24 h of the operation. Preoperative and postoperative diagnoses were determined based on the operative report. A negative laparotomy was defined as the lack of identifiable pathology during surgery as well as any indication for surgical repair. All paracenteses described were therapeutic paracenteses performed using a standard “Safe-T-Centesis” kit (CareFusion, Waukegan, IL).

Statistical Analysis

Descriptive statistics are reported as mean±standard deviation for normal distributions, median with interquartile range for nonparametric distributions, or as otherwise indicated. To examine factors associated with negative laparotomy, clinical, laboratory, and demographic data were first compared between patients with negative laparotomy to those with specific pathology identified in the operating room. Similarly, patient outcomes between those with positive and negative laparotomies were analyzed for statistical differences.

Next, the relationship between clinical factors and negative laparotomy was examined using univariate and multivariable logistic regression models. Therapeutic paracentesis within 48 h of surgery was used as our variable of interest, with covariates chosen based on biological plausibility for negative laparotomy and analyzed for the presence of confounding and collinearity. Similarly, the relationship between negative laparotomy and patient outcomes was explored in linear and logistic regression models using in-hospital mortality and 30-day readmission as binary outcomes and length of stay as a continuous variable. The association between negative laparotomy and mortality was examined in a multivariable model using previously validated predictors of mortality such as the MELD and ASA scores as covariates.^{7,9–12} Collinear variables were defined as those that altered the standard error of the variable of interest by greater than 10 % and were excluded from multivariable analyses as indicated. Comparisons of 30-day readmission were calculated excluding patients who died during the index hospitalization. For statistical comparisons, we used the chi-square test for binary and categorical variables, Student’s *t* test for normally distributed continuous variables, and Kruskal-Wallis test for continuous variables with nonparametric distribution. Data were analyzed using JMP SAS 11 pro© 2012 (SAS Institute Inc, Cary, NC, USA). Our protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki as reflected

in a priori approval as quality improvement by the institutional review board of the Beth Israel Deaconess Medical Center (Boston, MA).

Results

Patients and Population

Table 1 details the demographic and baseline clinical data of the 86 patients with cirrhosis who met the inclusion criteria for our study. Our cohort of cirrhotic patients who underwent emergent exploratory laparotomy represented a group at high risk for death, with an overall 90-day mortality rate of 39.5 % and in-hospital mortality of 33.7 %. Presumed diagnoses prompting operative intervention included perforated viscus (24.4 %), hemorrhage (15.1 %), bowel ischemia (19.8 %), bowel obstruction (26.7 %), cholecystitis (12.8 %), and compartment syndrome (1.2 %). Mean MELD score in our cohort was 21.3 ± 7.95 with ASA scores ranging from 3 (16.3 %) to 5 (8.1 %).

Patient Outcomes and Factors Associated with In-Hospital Mortality

Patient outcomes are summarized in Tables 1 and 2. In our cohort, patients had a median length of stay of 15 days (IQR 7–28.3) with a 30-day readmission rate amongst the survivors of 40.4 %. Compared to patients who had interventions at the time of operation (positive laparotomy), cirrhotic patients with negative laparotomy had a significantly higher in-hospital mortality (66.7 vs. 28.4 %; $P=0.018$).

In addition to having had a negative laparotomy, higher MELD score ($P<0.001$) and an ASA score >4 ($P=0.003$) were also significantly associated with higher in-hospital mortality, in a univariate analysis. When adjusted for MELD, negative laparotomy was an independent predictor of inhospital mortality (OR 4.7; $P=0.034$). However, when both MELD and ASA class >4 were included as covariates in a multivariable model, negative laparotomy was not a significant independent predictor of in-hospital mortality ($P=0.10$) (Table 3).

Factors Associated with a Negative Laparotomy

Out of the 86 cirrhotic patients included in our study who underwent nonhepatic emergent abdominal surgery, 12 (16.2 %) had no significant intraoperative findings and surgical repair was not performed (i.e., negative laparotomy) (Supplemental Table A). The factors that were associated with negative laparotomy within our cohort of patients who underwent emergent surgery are detailed in Table 4. For example, free air noted on CT imaging and a paracentesis performed 48 h prior to surgery were both significantly associated with having a negative laparotomy ($P=0.021$ and $P=0.016$, respectively). Interestingly, free air on CT imaging—always described in the radiology report as “greater than expected after a paracentesis”—was encountered more frequently in patients who had had a recent paracentesis, with 64.3 % (9/14) of those who had a paracentesis within the preceding 48 h noted to have substantial free air compared to 10.1 % (7/72) in patients in patients who did not undergo a paracentesis ($P<0.001$). A total of nine patients were noted to have substantial free air on CT following paracentesis, of which 5 (55.5 %) had negative findings at the time of emergent laparotomy. On the other hand, the rate of a negative laparotomy after the finding of free air on CT was nil in the population of cirrhotic patients who did not have a

paracentesis within 48 h of surgery ($P=0.038$). In those patients that did have a finding of perforation during surgery for free air following paracentesis, none were a complication of paracentesis (two with perforated diverticulitis; two with perforated ischemic colitis) (Table 5). Of note, none of the patients had ascetic fluid analysis consistent with infection or hemorrhage at the time of the procedure.

In a multivariable analysis, independent factors significantly associated with negative intraoperative findings during emergent surgery were (1) paracentesis prior to a preoperative diagnosis of perforated viscus ($P=0.006$), (2) developing an indication for emergent surgery greater than 24 h into hospital admission for another reason ($P=0.020$), and (3) a preoperative diagnosis of bowel ischemia ($P=0.005$), with odds ratios of 10.1, 5.80, and 11.1, respectively (Table 4).

Discussion

The goal of our study of cirrhotic patients who underwent emergent, nonhepatic abdominal surgery was to identify patients at risk for negative laparotomy. This study yielded three principle findings. First, we noted a significant rate of nonpathologic free intraperitoneal air following paracentesis. Second, negative laparotomy was associated with higher inhospital mortality. Third, the factors that were associated with a negative laparotomy included (a) recent paracentesis, (b) initial admission to a medical service and developing an indication for emergent surgery after more than 24 h in the hospital, and (c) suspected bowel ischemia. Consideration of these clinical features may be useful in the preoperative selection of appropriate surgical candidates.

Our findings regarding the association of paracentesis, intraperitoneal free air, and negative laparotomy are notable. Paracentesis is a common procedure for cirrhotic patients admitted to the hospital with ascites.¹⁵ Many patients must be evaluated for spontaneous bacterial peritonitis which can be present in many ways, including confusion, abdominal pain, fever, or renal dysfunction. Many other patients present with urgent discomfort related to ascites buildup requesting a therapeutic paracentesis, often in the setting of progressive renal insufficiency.^{16,17} Furthermore, patients with decompensated cirrhosis admitted for management of spontaneous bacterial peritonitis, hepatic encephalopathy, gastrointestinal bleeding, or acute renal failure are all at high risk for death and often present with higher MELD score and ASA class, both of which powerfully predict overall and postsurgical mortality alike.^{10–12,18–20}

These patients with cirrhosis are at significant risk to develop progressive acute-on-chronic liver failure over the course of hospitalization despite optimal medical therapy. In this context, following a paracentesis, it may be difficult to discern whether a patient's decompensation is related to a procedural complication or the worsening of an underlying medical condition. As evidenced by our data, free air on CT does not reliably indicate the presence of clinically important perforation in patients who have undergone recent paracentesis. Beyond that, we found that being initially admitted to a medical service and subsequently developing an indication for emergent surgery after more than 24 h of admission to the hospital were also significantly associated with negative laparotomy.

Furthermore, negative laparotomy is associated with mortality adjusting for MELD but not when one accounts for ASA class. All of these factors are consistent with the conclusion that negative laparotomy is more likely to be encountered in decompensated, sick patients with medical conditions refractory to therapy, who develop their indications for surgery as established inpatients.

The cause of nonpathologic pneumoperitoneum following paracentesis is not entirely clear. It may be related to procedural technique, equipment used, or clinically insignificant microscopic perforation. Nonpathologic free air has been described following percutaneous endoscopic gastrostomy (PEG) tube placement as well as in patients undergoing peritoneal dialysis and, now, after paracentesis.^{21–25} When free air is detected within 48 h after a paracentesis, without other obvious pathology such as diverticulitis, our data suggests that consideration should be given to close clinical observation rather than surgery. A strategy of supportive care and careful, ultrasound-guided diagnostic paracenteses to identify secondary bacterial peritonitis as an objective parameter of a perforated viscus and intraabdominal sepsis may increase operative yield.^{26,27}

A preoperative diagnosis of bowel ischemia was also associated with higher odds of negative laparotomy. In retrospect, it appears that these patients had refractory sepsis and portal hypertension leading to the constellation of bowel congestion, shock, and rising lactate. This highlights a diagnostic dilemma in the context of decompensated cirrhosis. Mucosal edema on CT may reflect ischemia, but patients with cirrhosis often have bowel wall edema secondary to portal hypertension.²⁸ Additionally, while the clinical picture of ischemic bowel includes refractory shock and lactic acidosis, patients with cirrhosis have reduced lactate clearance in general and especially in the setting of sepsis.²⁹ Therefore, further study to optimize the preoperative diagnosis and CT performance for diagnosing bowel ischemia in decompensated cirrhotic patients may prove useful.

Independent of preoperative diagnosis, negative laparotomy is associated with in-hospital mortality. Our data suggests two explanations. First, surgery may be performed on decompensating patients after all other medical interventions are exhausted. Second, negative laparotomy is by definition noncorrective surgery that adds morbidity to the hospital course of an otherwise decompensated patient.

Our study has limits. First, it is retrospective, and therefore, we cannot fully account for confounders unavailable in the electronic medical record. Second, we studied a cohort of patients with cirrhosis who underwent emergent laparotomy, all with an ASA class of 3 or greater. As such, our results are not generalizable to stable cirrhotic patients undergoing elective surgery. Third, some of our associations may be related to institution-specific features including the kit employed for therapeutic paracenteses and the preoperative radiographic interpretations. Additionally, we do not know the true rate of free intraperitoneal air following paracentesis in patients with cirrhosis who did not undergo cross-sectional imaging. This requires further study. Fourth, robust identification of predictors for negative laparotomy is limited by the sample size of patients who had negative findings during surgery. Additionally, since our study included only patients who underwent emergent surgery, we cannot comment on the outcomes of patients discovered to have

intraoperative free air following paracentesis but of patients who were managed expectantly without surgery. This is an area for future investigation. Finally, we must acknowledge the difficulty of the clinical decision making as experienced by our surgeons when faced with the hope of a reversible cause of decompensation in desperately ill patients. There are scenarios for these sick and vulnerable patients where surgical intervention has proven effective and life saving.³⁰ The rationale for this research is to help determine those scenarios where it is not.

In summary, paracentesis in cirrhotic patients is associated with nonpathologic free air and emergent surgery for repair of perforated viscus in these patients may not be warranted. Emergent noncorrective surgery in these patients is associated with higher in-hospital mortality. In order to improve the care of patients with decompensated cirrhosis with suspected acute abdomen, investigation with additional preoperative diagnostics, such as repeat paracentesis assessing for secondary bacterial peritonitis, should be considered for selected patients with risk factors for negative laparotomy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

1. Lindenmuth WW, Eisenberg MM. The surgical risk in cirrhosis of the liver. Arch Surg. 1963; 86:235–42. [PubMed: 13930804]
2. Aranha GV, Greenlee HB. Intra-abdominal Surgery in Patients With Advanced Cirrhosis. Arch Surg. 1986; 121:275–277. [PubMed: 3947226]
3. Greenwood SM, Leffler CT, Minkowitz S. The increased mortality rate of open liver biopsy in alcoholic hepatitis. SurgGynecol Obstet. 1972; 134(4):600–4.
4. Csikesz NG, Nguyen LN, Tseng JF, Shah SA. Nationwide volume and mortality after elective surgery in cirrhotic patients. J Am Coll Surg. 2009; 208(1):96–103. [PubMed: 19228510]
5. Mansour A, Watson W, Shayani V, Pickleman J. Abdominal operations in patients with cirrhosis: still a major surgical challenge. Surgery. 1997; 122:730–735. [PubMed: 9347849]
6. Powell-Jackson P, Greenway B, Williams R. Adverse effects of exploratory laparotomy in patients with unsuspected liver disease. Br J Surg. 1982; 69:449–451. [PubMed: 7104630]
7. Befeler AS, Palmer DE, Hoffman M, Longo W, Solomon H, Di Bisceglie AM. The safety of intra-abdominal surgery in patients with cirrhosis: model for end-stage liver disease score is superior to Child-Turcotte-Pugh classification in predicting outcome. Arch Surg. 2005; 140(7):650–4. [PubMed: 16027329]
8. Friedman LS. The risk of surgery in patients with liver disease. Hepatology. 1999; 29(6):1617–23. [PubMed: 10347099]
9. Teh SH, Nagorney DM, Stevens SR, Offord KP, Therneau TM, Plevak DJ, et al. Risk factors for mortality after surgery in patients with cirrhosis. Gastroenterology. 2007; 132(4):1261–9. [PubMed: 17408652]
10. Telem DA, Schiano T, Goldstone R, Han DK, Buch KE, Chin EH, et al. Factors that predict outcome of abdominal operations in patients with advanced cirrhosis. Clin Gastroenterol Hepatol. 2010; 8(5):451–7. [PubMed: 20036761]
11. Ziser A, Plevak DJ, Wiesner RH, Rakela J, Offord KP, Brown DL. Morbidity and mortality in cirrhotic patients undergoing anesthesia and surgery. Anesthesiology. 1999; 90:42–53. [PubMed: 9915311]

12. Kim SH, Han YD, Lee JG, Kim do Y, Choi SB, Choi GH, et al. MELD-based indices as predictors of mortality in chronic liver disease patients who undergo emergency surgery with general anesthesia. *J Gastrointest Surg.* 2011; 15:2029–2035. [PubMed: 21913042]
13. Neeff H, Mariaskin D, Spangenberg HC, Hopt UT, Makowiec F. Perioperative mortality after non-hepatic general surgery in patients with liver cirrhosis: an analysis of 138 operations in the 2000s using Child and MELD scores. *J Gastrointest Surg.* 2011; 15:1–11. [PubMed: 21061184]
14. Garrison RN, Cryer HM, Howard DA, Polk HC Jr. Clarification of risk factors for abdominal operations in patients with hepatic cirrhosis. *Ann Surg.* 1984; 199(6):648–55. [PubMed: 6732310]
15. Runyon BA. Management of adult patients with ascites due to cirrhosis: An update. *Hepatology.* 2009; 49(6):2087–2107. [PubMed: 19475696]
16. Moreau R, Jalan R, Ginès P, Pavesi M, Angeli P, Cordoba J, et al. Acute on Chronic Liver Failure definition. *Gastroenterology.* 2013; 144(7):1426–1437. [PubMed: 23474284]
17. Wong F, O’Leary JG, Reddy KR, Patton H, Kamath PS, Fallon MB, et al. New Consensus Definition of Acute Kidney Injury Accurately Predicts 30-Day Mortality in Patients With Cirrhosis and Infection. *Gastroenterology.* 2013; 145(6):1280–8. [PubMed: 23999172]
18. Bajaj JS, O’Leary JG, Reddy KR, Wong F, Olson JC, Subramanian RM, et al. Second infections independently increase mortality in hospitalized patients with cirrhosis: the North American consortium for the study of end-stage liver disease (NACSELD) experience. *Hepatology.* 2012; 56(6):2328–2335. DOI: 10.1002/hep.25947 [PubMed: 22806618]
19. Bustamante J, Rimola A, Ventura PJ, Navasa M, Cirera I, Reggiardo V, Rodés J. Prognostic significance of hepatic encephalopathy in patients with cirrhosis. *J Hepatol.* 1999; 30(5):890–895. [PubMed: 10365817]
20. del Olmo JA, Peña A, Serra MA, Wassel AH, Benages A, Rodrigo JM. Predictors of morbidity and mortality after the first episode of upper gastrointestinal bleeding in liver cirrhosis. *J Hepatol.* 2000; 32(1):19–24.
21. Mularski RA, Sippel JM, Osborne ML. Pneumoperitoneum: a review of nonsurgical causes. *Crit Care Med.* 2000; 28(7):2638–2644. [PubMed: 10921609]
22. Kiefer T, Schenk U, Weber J, Hübel E, Kuhlmann U. Incidence and significance of pneumoperitoneum in continuous ambulatory peritoneal dialysis. *Am J Kidney Dis.* 1993; 22(1):30–35. [PubMed: 8322790]
23. Wiesen AJ, Sideridis K, Fernandes A, Hines J, Indaram A, Weinstein L, et al. True incidence and clinical significance of pneumoperitoneum after PEG placement: a prospective study. *YMGE.* 2006; 64(6):886–889.
24. Blum CA, Selander C, Ruddy JM, Leon S. The incidence and clinical significance of pneumoperitoneum after percutaneous endoscopic gastrostomy: a review of 722 cases. *Am Surg.* 2009; 75(1):39–43. [PubMed: 19213395]
25. Gottfried EB, Plumser AB, Clair MR. Pneumoperitoneum following percutaneous endoscopic gastrostomy. A prospective study. *YMGE.* 1986; 32(6):397–399.
26. Imai K, Doi Y, Takata N, Yoshinaka I, Harada K. Successful conservative treatment of pneumatosis intestinalis associated with intraperitoneal free air: report of a case. *Surg Today.* 2012; 42(10):992–996. [PubMed: 22699401]
27. Soriano G, Castellote J, Alvarez C, Girbau A, Gordillo J, Baliellas C, et al. Secondary bacterial peritonitis in cirrhosis: a retrospective study of clinical and analytical characteristics, diagnosis and management. *J Hepatol.* 2010; 52(1):39–44. [PubMed: 19897273]
28. Takahashi Y, Fujimori S, Narahara Y, et al. Small intestinal edema had the strongest correlation with portal venous pressure amongst capsule endoscopy findings. *Digestion.* 2012; 86(1):48–54. [PubMed: 22710440]
29. Andersen LW, Mackenhauer J, Roberts JC, Berg KM, Cocchi MN, Donnino MW. Etiology and therapeutic approach to elevated lactate levels. *Mayo Clin Proc.* 2013; 88(10):1127–40. [PubMed: 24079682]
30. Orloff MJ, Isenberg JI, Wheeler HO, et al. Emergency portacaval shunt versus rescue portacaval shunt in a randomized controlled trial of emergency treatment of acutely bleeding esophageal varices in cirrhosis—part 3. *J Gastrointest Surg.* 2010; 14(11):1782–95. [PubMed: 20658205]

Table 1

Baseline characteristics of 86 patients with cirrhosis who underwent emergent surgery

Number	86
Age (years; mean±SD)	58.43±10.73
Male gender (<i>N</i> ; %)	57 (66.3)
ASA class (<i>N</i> ; %)	
3	14 (16.3)
4	65 (75.6)
5	7 (8.1)
MELD (mean±SD)	21.3±7.95
Admission service (<i>n</i> ; %)	
Medical	25 (29.1)
Surgical	45 (52.3)
Intensive care unit	16 (18.6)
Preoperative diagnosis	
Perforated viscus	21 (24.4)
Hemorrhage	13 (15.1)
Bowel ischemia	17 (19.8)
Bowel obstruction	23 (26.7)
Cholecystitis	11 (12.8)
Compartment syndrome	1 (1.2)
Patients with negative operative findings at laparotomy, i.e., negative laparotomy (<i>N</i> ; %)	12 (16.2)
Patients with >1 day spent in ICU (<i>N</i> ; %)	62 (72)
LOS (days; mean±SD; median (IQR))	20.2±17.4; 15 (7–28.3)
ICU LOS (mean±SD; median (IQR))	11.8±10.5; 9 (4–18)
30-day readmission rate (<i>n</i> ; %)	23 (40.4)
In-hospital mortality (<i>n</i> ; %)	29 (33.7)

ASA American Society of Anesthesiologists, *ICU* intensive care unit, *IQR* interquartile range, *LOS* length of stay, *MELD* Model for End-Stage Liver Disease, *SD* standard deviation

Table 2

Relationship of operative findings with patient outcomes

	Positive laparotomy	Negative laparotomy	<i>P</i> value
LOS (mean±SD; median (IQR))	19.4±17.7; 12.5 (6.75–28)	24.8±15.5; 26 (13.5–32.5)	0.28
ICU LOS	11.7±10.9; 8 (4–16)	12±8.9; 11 (4–18)	0.9
30-day readmission rate (<i>N</i> ; %)	21 (39.6)	2 (50)	0.68
In-hospital mortality (<i>N</i> ; %)	21 (28.4)	8 (66.7)	0.02

ICU intensive care unit, *IQR* interquartile range, *LOS* length of stay, *SD* standard deviation

Table 3

Factors associated with in-hospital mortality

	Univariate (OR)	P value	Multivariate (OR)	P value
Age	1.02	0.27	1.03	0.26
Gender	1.53	0.39	0.91	0.88
Negative laparotomy	5.04	0.01	3.36	0.1
MELD	1.20	0.001	1.21	0.001
ASA class >4	14.6	0.003	13.2	0.01

Multivariate analysis including age, gender, negative laparotomy, MELD, and ASA class

ASA American Society of Anesthesiologists, MELD Model for End-Stage Liver Disease, OR odds ratio

Table 4

Factors associated with negative laparotomy

	Positive laparotomy	Negative laparotomy	Univariate regression (OR)	Multivariate regression ^a (OR)
Number	74	12		
Age (mean±SD)	58.7±10.6	57.1±11.8	0.99 <i>P</i> =0.6	0.97 <i>P</i> =0.45
Male gender (<i>N</i> ; %)	48 (64)	9 (75)	1.63 <i>P</i> =0.5	2.5 <i>P</i> =0.26
Preoperative Diagnosis				
Perforated viscus	14 (18.9)	7 (58.3)	6.0 <i>P</i> =0.006	
Bowel ischemia	12 (16.2)	5 (41.7)	3.69 <i>P</i> =0.046	11.1 <i>P</i> =0.005
Other	48 (64.8)	0 (0)	0.01 <i>P</i> =0.001	
Paracentesis 48 h prior to surgery	9 (12.2)	5 (41.7)	5.16 <i>P</i> =0.02	10.1 <i>P</i> =0.006
Free air on CT	10 (15.9)	5 (50)	5.30 <i>P</i> =0.02	
Admission service				
Medicine	18 (24.3)	7 (58.3)	4.36 <i>P</i> =0.02	
Surgery or ICU	66 (75.7)	5 (41.6)	0.23 <i>P</i> =0.02	
Surgery >24 h after admission	23 (31)	8 (66.7)	4.43 <i>P</i> =0.02	5.80 <i>P</i> =0.02

CT computed tomography, *ICU* intensive care unit, *OR* odds ratio, *SD* standard deviation, *SNF* skilled nursing facility

^a Covariates included age, gender, ischemic injury, and paracentesis within 48 h of surgery and surgery >24 h after admission. Covariates perforated viscus, other surgery, free air on CT, and admission to medicine service were excluded due to collinearity

Table 5

Postoperative diagnoses in 14 patients who underwent emergent surgery 48 h after paracentesis

Preoperative diagnosis	Intraoperative findings	Postoperative diagnosis	Complication of paracentesis
Perforation (based on free air) (<i>N</i> =9)	No abnormal finding (<i>N</i> =5)	No abnormal finding (<i>N</i> =4)	None
		Sepsis (<i>N</i> =1)	
	Bowel ischemia (<i>N</i> =1)	Bowel ischemia (<i>N</i> =2)	
	Colonic perforation (<i>N</i> =3)	Perforated diverticulitis (<i>N</i> =2)	
Bowel ischemia (<i>N</i> =1)	Bowel ischemia (<i>N</i> =1)	Bowel ischemia (<i>N</i> =1)	
Hemorrhage (<i>N</i> =4)	Hemoperitoneum (<i>N</i> =3)	Paracentesis-related hemoperitoneum (<i>N</i> =2)	Hemoperitoneum (<i>N</i> =2)
		Spontaneous hemoperitoneum due to variceal rupture (<i>N</i> =1)	
	Abdominal wall hematoma (<i>N</i> =1)	Paracentesis-related abdominal wall hematoma (<i>N</i> =1)	Abdominal wall hematoma (<i>N</i> =1)