Liver Cirrhosis: An Unfavorable Factor for Nonoperative Management of Blunt Splenic Injury

Jen-Feng Fang, MD, Ray-Jade Chen, MD, Being-Chuan Lin, MD, Yu-Bau Hsu, MD, Jung-Liang Kao, MD, and Miin-Fu Chen, MD

Background: **Nonoperative management (NOM) of blunt splenic injury (BSI) is currently a well-accepted treatment modality for hemodynamically stable patients. More than 60% of BSI patients can be successfully treated without operation. Old age, high-grade injury, contrast blush, and multiple associated injuries were reported to have a higher failure rate but not to be exclusive of NOM. The purpose of this study was to review the treatment courses and results of a special group of BSI patients with coexistent liver cirrhosis. Factors leading to poor results were analyzed and treatment strategy was proposed accordingly.**

Methods: **During a 5-year period, 487 patients with BSI were treated following a standard protocol. Twelve of them had underlying liver cirrhosis. The medical records, radiographic findings, laboratory data, and operative variables were retrospectively reviewed.**

Results: **Eighty-nine (18%) patients had immediate celiotomy for splenic hemorrhage with unstable hemodynamic status, 59 (12%) had non–spleen-related or nontherapeutic laparotomy, and 339 (70%) patients received NOM initially. Failure of NOM was found in 74 patients (22%). Twelve patients with initial NOM had coexistent liver cirrhosis. The amount of blood transfusion within 72 hours after admission for these 12 patients ranged from 4 to 26 units. Patients with coexistent liver cirrhosis and BSI had a significantly higher NOM failure rate (92% vs. 19%). In NOM failure patients, those with liver cirrhosis had lower Injury Severity Scores, lower splenic injury severity grades, more blood transfusions, and a higher mortality rate. Risk factors for mortality in these patients included a higher Injury Severity Score, a severely elevated prothrombin time (PT), a larger transfusion requirement, and a lower serum albumin level.**

Conclusion: **Liver cirrhosis with subsequent development of portal hypertension, splenomegaly, and coagulopathy makes spontaneous hemostasis of the injured spleen difficult. NOM for BSI patients with coexistent liver cirrhosis carries a high failure and mortality rate. NOM may be successful in only a small group of patients with low-grade single-organ injury and with a normal or mildly elevated PT. Aggressive correction of coagulopathy should be performed in these patients. High-grade splenic injury, multiple associated injuries, and an elevated PT are indicators for early surgery. The mortality rate is high in patients with a severely prolonged PT irrespective of treatment modalities.**

Key Words: **Blunt splenic injury (BSI), Liver cirrhosis, Nonoperative management (NOM), Prothrombin time (PT).**

J Trauma. 2003;54:1131–1136.

Nonoperative management (NOM) is widely accepted as a safe and effective treatment modality for hemodynamically stable patients with blunt splenic injury (BSI). NOM is currently used in up to 70% of patients with BSI. With increasing experience, the inclusion criteria of NOM have been extended to those patients who would have been operated on in the past decade. High-grade splenic injury, large amount of hemoperitoneum, associated neurologic injury, and old age have been reported to be associated with increased risk of failure of NOM.[1–4] However, approximately 80% of these high-risk patients have been successfully managed nonoperatively. These factors are, therefore, not considered to be absolute contraindications for NOM of BSI patients.[1–4] Pachter et al. even reported successful application of NOM to 15 patients with preexisting splenic abnormality from a variety of diseases.[5] As a result, aggressive NOM has been applied to most patients with BSI and to some selected patients with stab wounds. The only universally accepted absolute criterion for surgical intervention is the presence of hemodynamic instability.

Aggressive application of NOM to BSI patients carries some potential risks. Cocanour et al. reported that seven patients (8%) managed nonoperatively developed delayed splenic complications including overt bleeding or abscess formation. They claimed that some complications were life threatening and might require surgical intervention.[6] Early identification of patients with high risk of NOM failure may prevent these complications from happening. Grade III or higher splenic injury and transfusion of more than one unit of blood were identified as two independent risk factors for failure of NOM by Velmahos et al. in a retrospective study.[7] The coexistence of both risk factors predicted failure of NOM in 97% of patients. Lowering the threshold for operation or intensifying the monitoring is highly recommended for such patients.[7]

Submitted for publication May 27, 2002.
Accepted for publication January 30, 2003.

From the Department of Surgery, Divisions of Trauma and Emergency Surgery (J.-F.F., R.-J.C., B.-C.L., Y.-B.H., J.-L.K.) and General Surgery (M.-F.C.), Chang Gung Memorial Hospital, Chang Gung University, Kweishan, Taoyuan, Taiwan.

Address for reprints: Jen-Feng Fang, MD, Department of Surgery, First Division of Trauma and Emergency Surgery, Chang Gung Memorial Hospital, Chang Gung University, 5, Fushing Street, Kweishan, Taoyuan, Taiwan; email: jimfang@adm.cgmh.org.tw.

DOI: 10.1097/01.TA.0000066123.32997.BB

Liver cirrhosis is a common disease in Taiwan. It is most frequently associated with hepatitis B or C infection. Because approximately 75% of the world's hepatitis B carriers live in Asian countries,[8] it is not unusual to encounter BSI patients with underlying liver cirrhosis in these countries. Liver cirrhosis is more frequently associated with alcohol ingestion in Western countries. Up to 15% of chronic alcoholics develop liver diseases or cirrhosis.[9] Alcoholic patients are more susceptible to trauma. BSI in patients with liver cirrhosis has special problems. Many investigators have reported that patients with liver cirrhosis tend to have grave postoperative complication after abdominal operations, frequently leading to their eventual death.[10–14] From this point of view, NOM of splenic injuries would be the treatment of choice for these patients. Liver cirrhosis, however, results in subsequent portal hypertension, splenomegaly, and deficiency of coagulation factors. Spontaneous hemostasis of the injured spleen may be interfered with by these changes and NOM may not be successful. The outcome and policy of treatment for BSI patients with liver cirrhosis thus need to be further clarified. The objectives of this study were to review the treatment courses and results of a special group of BSI patients with underlying liver cirrhosis, to compare the characteristics and causes of failure of NOM between patients without (control group) and with liver cirrhosis, and to analyze risk factors leading to poor results in liver cirrhotic patients and propose treatment strategy accordingly.

PATIENTS AND METHODS

During a 5-year period from January 1997 to December 2001, 487 patients with BSI were admitted to the Department of Trauma and Emergency Surgery, Chang Gung Memorial Hospital. Standard protocols of resuscitation established by the American College of Surgeons Committee on Trauma were used. Patients with hemodynamic instability unresponsive to fluid resuscitation and two units of blood transfusion would receive emergent celiotomy. Patients who had hollow organ injury diagnosed by frank peritoneal signs or diagnostic peritoneal lavage would also receive emergent celiotomy.[15] Stable initial hemodynamic status (systolic blood pressure maintained at $>$ 90 mm Hg for more than 30 minutes) or stable hemodynamic status achieved with less than two units of blood transfusion qualified the patients for nonoperative management. These patients would receive abdominal computed tomographic (CT) examination at emergency service and be admitted to the intensive care unit for close monitoring of hemodynamic status, hematocrit levels, peritoneal signs, and intra-abdominal pressures. NOM would be terminated and celiotomy would be performed in patients who had ongoing hemorrhage requiring more than four units of spleen-related blood transfusion to maintain hemodynamic stability within 72 hours, delayed hemorrhage and unstable hemodynamic status requiring more than four units of blood transfusion, and an intra-abdominal pressure higher than 30 cm H_2O.

The medical records, trauma registry records, radiologic findings, and laboratory data of all patients were reviewed. The patient's age, gender, mechanism of injury, Injury Severity Score (ISS), associated injuries, treatment, interval between injury and operation if celiotomy was performed, laboratory data, amount of blood transfusion within 72 hours after admission, length of intensive care unit and hospital stays, overall hospital course, and clinical outcome were recorded.

The patients who received celiotomy were classified as having a spleen-related celiotomy if active bleeding from the spleen was found and some procedures to stop the bleeding were required. The patients were classified as having a non–spleen-related celiotomy if there was no active bleeding from the spleen and procedures for splenic hemorrhage control were not required but procedures for other intra-abdominal injuries were needed during operation. The operation was classified as nontherapeutic if no specific procedure was required for the intra-abdominal injury. Failure of NOM was defined as the necessity of operation for the spleen after a period of NOM.

Special attention was paid to patients with failure of NOM. These patients were divided into two groups according to the presence or absence of liver cirrhosis. The clinical variables, radiologic grading, prothrombin time (PT), serum albumin level, serum bilirubin level, serum alkaline phosphatase level, amount of blood transfusion, and outcomes were compared. Risk factors leading to mortality in liver cirrhotic patients were also analyzed.

For NOM failure patients, several variables were compared between liver-cirrhotic and non–liver-cirrhotic patients. Nonparametric data were evaluated with Fisher's exact test, ordinal values with Wilcoxon rank sum test, and continuous values by *t* test. To evaluate the risk factors affecting the outcome of patients with concurrent BSI and liver cirrhosis, contingency tables were created for injury and hepatic parameters as related to survival and subjected to Fisher's exact test for nonparametric data, Wilcoxon rank sum test for ordinal variables, and *t* test for continuous values. Statistical significance was defined as $p < 0.05$ for each test.

RESULTS

Of the 487 patients, 148 received emergent celiotomy initially. The operation was spleen-related in 89 patients, non–spleen-related in another 53 patients, and nontherapeutic in the other 6 patients. Three hundred thirty-nine patients had stable hemodynamic status and were treated nonoperatively. Seventy-four patients were diagnosed as having failure of NOM at an average of 49 hours after trauma. The reasons for termination of NOM were ongoing hemorrhage in 52 patients, delayed hemorrhage in 14 patients, and increased intra-abdominal pressure in 8 patients. Eleven of these patients had preexisting liver cirrhosis. There were 32 deaths in the 487 patients reviewed. Twenty-six patients died of severe multiple injuries. The causes of death of the other six patients were attributed to the splenic injury. These six patients all had coexistent liver cirrhosis.

Twelve patients in this series had evidence of liver cirrhosis based on clinical, pathologic, or radiologic grounds. Ten patients had been diagnosed as having liver cirrhosis

Table 1 **Demographic Data of Patients with Liver Cirrhosis and Blunt Splenic Injury**

Patient No.	Age (yr)	Sex	Mechanism	Injury Grade	ISS	PT (s)	BT (U)	IS (days)	HS (days)	Interval Inj-Op	Outcome
1	67	M	Fall#	III	9	16/11	6	1	20	7 days	Alive
2	67	F	Fall	II	4	16/11	6	1	8	10 days	Alive
3	26	M	MCC	III	9	NA	6	1	9	10 h	Alive
4	71	F	Fall#	II	4	16/11	6	2	20	3 days	Alive
5	49	M	Fall*	III	25	28/12	19	3	3	4 h	Dead
6	51	M	MVC	II	4	>100	20	1	1	8 h	Dead
7	37	M	MVC	III	18	NA	14	5	5	2 days	Dead
8	71	F	Fall#	II	4	18/11	14	10	17	7 h	Alive
9	65	F	MVC	III	25	>100	20	1	1	6 h	Dead
10	33	M	MCC	IV	16	>100	26	1	1	3 h	Dead
11	67	F	Ped	II	8	>100	12	3	24	6 h	Dead
12	36	M	Fall#	II	4	13/11	4	5	40	NA	Alive

BT, blood transfusion within 72 h; IS, ICU stay; HS, hospital stay; Inj-Op, injury to operation; MCC, motorcycle crash; MVC, motor vehicle crash; Ped, pedestrian; NA, not available.

* Fall from 6 m high; # fall down while walking.

before splenic trauma and were regularly followed. The remaining two patients had the diagnosis made during exploratory laparotomy for abdominal trauma. The cause of liver cirrhosis was attributed to alcohol ingestion in five patients and to previous hepatitis in the other seven patients.

The demographic data and outcomes of these 12 patients are shown in Table 1. Nine patients visited our emergency service soon after injury. The other three patients had delayed clinical presentation and sought medical help at 3, 7, and 10 days after the trauma separately. Seven patients were normotensive and five patients were hypotensive when they arrived at our emergency room. These twelve patients all had good response to initial resuscitation. Abdominal CT examinations were performed and the Injury Severity Scores were calculated to range from 4 to 25. Four patients had an ISS higher than 16. Five patients had associated injuries, which included two head injuries, one rib fracture, one maxillary fracture, and one renal injury. The distribution of splenic injury severity grade was as follows: grade II in six patients, grade III in five patients, and grade IV in one patient. Five patients had severely elevated PT (more than two times the level of the controls). Another five patients had mildly to moderately elevated PT (prolonged, but less than two times the level of the controls). Because history of liver cirrhosis had not been traced, PT was not checked preoperatively in the other two patients.

These 12 patients all received NOM initially. Ten patients showed deterioration of hemodynamic status within 1 to 12 hours after abdominal CT scan (at 3 hours–10 days after injury) and responded unsatisfactorily to four units of blood transfusion. NOM was terminated and emergent celiotomy with splenectomy was performed for these 10 patients. One patient developed abdominal compartment syndrome after receiving more than four units of blood transfusion within 30 hours. Laparotomy and splenectomy was performed for this patient at 32 hours after CT scan (35 hours after injury). The last patient was successfully treated with NOM.

For the 12 patients with liver cirrhosis, the amount of blood transfusion within 72 hours after admission ranged from 4 to 26 units. Six patients died. The mortality rate was 50%. Three of these six patients died within 24 hours after operation. The other three patients died at 3 to 24 days after operation. Two patients had grade II, three had grade III, and one had grade IV splenic injuries. All except one (in whom liver cirrhosis was not diagnosed preoperatively) had severely prolonged PTs before operation. Active bleeding from the spleen and massive hemoperitoneum were noted during operation. They developed multiple organ system dysfunction during or soon after operation, which responded poorly to aggressive resuscitation and intensive care.

Six patients survived. Five of them had mild to moderate elevation of PT. The last one did not have PT checked because liver cirrhosis had not been diagnosed before this injury. Four patients had grade II and two patients had grade III splenic injuries. Five of them required laparotomy and splenectomy at 7 hours to 10 days after injury. One patient developed multiple organ dysfunction syndrome later and had good response to intensive care. The other four patients recovered smoothly after operation. NOM was successfully applied to one patient with grade II splenic injury. This patient required four units of blood transfusion. The clinical course was complicated with hepatic failure, requiring prolonged hospitalization.

Comparison of NOM Failure between Patients with or without Liver Cirrhosis

A total of 74 patients had failure of NOM. They were divided into two groups according to the presence or absence of liver cirrhosis. Patients with liver cirrhosis had a significantly higher chance of NOM failure ($p < 0.001$). Eleven of 12 (92%) BSI patients with coexistent liver cirrhosis had failure of NOM, whereas 63 of 327 (19%) BSI patients without coexistent liver cirrhosis had failure of NOM. The demographic data, injury

Table 2 Comparison of Demographic Data and Characteristics of NOM Failed Patients with or without Liver Cirrhosis

	Patients without Liver Cirrhosis	Patients with Liver Cirrhosis	*p* Value
Age (yr)	17–75 (51 ± 16)	26–71 (55 ± 17)	NS
Sex	M:F = 41:22	M:F = 6:5	NS
ISS	9–36 (median, 16)	4–25 (median, 8.5)	0.006
Injury grade	II:III:IV:V = 0:18:39:6	II:III:IV:V = 5:5:1:0	<0.001
Elevated PT	—	Severe:moderate = 5:4 (2 unchecked)	NA
Amount of BT (U)	5–10 (6.3 ± 1.6)	6–26 (13.5 ± 7.1)	0.006
Interval Inj-Op	3 h–18 days (48 ± 62 h)	3 h–10 days (52 ± 56 h)	NS
ICU stay (days)	1–6 (1.8 ± 2.4)	1–10 (2.6 ± 2.7)	NS
Hospital stay (days)	6–17 (8.5 ± 5.2)	1–24 (12.5 ± 12.1)	NS
Complication	7/63	3/11	NS
Mortality	2*/63	6/11 (55%)	<0.001

NS, not significant; NA, not applicable; ICU, intensive care unit.
* Both related to severe head injury.

characteristics, and hepatic parameters are shown in Table 2. Non–liver-cirrhotic patients had significantly higher Injury Severity Scores, higher injury severity grades of the spleen, and fewer blood transfusions. Two patients in the non–liver-cirrhotic group died of severe head injury. Liver-cirrhotic patient also demonstrated a significantly higher spleen-related mortality rate (6 of 11 vs. 0 of 63, $p < 0.001$).

Risk Factor Analysis for Mortality of BSI Patients with Coexistent Liver Cirrhosis

There were 32 deaths in the entire group. Twenty-six patients died of associated injuries and six patients died of splenic injury. All these six patients had coexistent liver cirrhosis. In patients with coexistence of liver cirrhosis and BSI, 50% died despite emergent splenectomy, aggressive replacement of coagulation factors, and blood transfusion. The demographic data, injury characteristics, and hepatic parameters are shown in Table 3. The mortality patients had higher Injury Severity Scores, more blood transfusions, more severely elevated PTs, and lower serum albumin levels. There were no differences in the mechanism of injury, age, sex, presence of encephalopathy, and serum bilirubin levels.

DISCUSSION

NOM is currently applicable to more than 60% of patients with BSI.[5,16,17] The successful rate of NOM is reported to be up to 90%.[1–5] NOM is especially valuable in patients with low-grade splenic injuries. Some studies have reported that NOM fails only in patients sustaining high-grade (grade III–V) injuries.[1,5,7] This treatment modality can even be applied to patients with preexisting splenic abnormalities.[5] In the present study, NOM has been initially applied to 339 (70%) of 487 BSI patients, with 265 (78%) of them being successfully treated. The failure rate of NOM in our series is 21.9%, which is high compared with the reported data. This may be attributed to the low threshold of including BSI patients for NOM in our institute. Using unstable hemodynamic status as a single indicator for operation may delay the treatment of some patients who eventually will need operation. Searching for possible preexisting conditions and variables not suitable for NOM is thus necessary.

In our series, the patients with preexistent liver cirrhosis have a significantly higher NOM failure rate (92%) when compared with that (19%) of noncirrhotic patients. For the

Table 3 Risk Factor Analysis for Mortality in Patients with Coexistent Liver Cirrhosis and Blunt Splenic Injury

	Nonsurvivors	Survivors	*p* Value
Age (yr)	33–67 (50 ± 14)	26–71 (56 ± 20)	NS
Sex	M:F = 4:2	M:F = 3:3	NS
ISS	4–25 (median, 17)	4–9 (median, 4)	0.045
Injury grade	II, 2; III, 3; IV, 1	II, 4; III, 2	NS
Amount of BT (U)	12–26 (18.5 ± 5.0)	4–14 (7 ± 3.5)	<0.001
Interval Inj-Op	3 h–2 days (13 ± 17h)	7 h–10 days (99 ± 102h)	NS
Encephalopathy	Absent	Absent	NA
Serum albumin (g/dL)	1.8–2.9 (2.3 ± 0.4)	2.5–3.3 (2.9 ± 0.3)	0.006
Serum bilirubin (mg/dL)	1.2–2.5 (1.9 ± 0.3)	1.2–3.8 (2.2 ± 0.5)	NS
SGOT (U/L)	39–145 (61 ± 25)	45–156 (65 ± 28)	NS
PT (s)	28–100 (85 ± 32)	13–18 (15.8 ± 1.8)	<0.001
BUN (mg/dL)	9–26 (19 ± 8.5)	18–25 (21 ± 2.8)	NS

NS, not significant; NA, not applicable; SGOT, serum glutamic oxaloacetic transaminase; BUN, blood urea nitrogen.

NOM failure patients, those who have liver cirrhosis are less severely injured, as shown by lower ISS and lower injury severity grades of the spleen. These patients, however, need more blood transfusions and have a higher mortality rate. These results are very different from what have been observed in patients without coexistent liver cirrhosis and make BSI in patients with coexistent liver cirrhosis a special condition necessitating further investigation.

The reasons for the high failure rate of NOM in patients with coexistent BSI and liver cirrhosis have rarely been discussed in the English literature. Spontaneous hemostasis seems to be difficult for these patients. We hypothesize that it may be the result of the pathophysiologic changes induced by liver cirrhosis. Most patients with liver cirrhosis will develop subsequent portal hypertension and splenomegaly. Enlarged spleen alone, as reported by Pachter et al.,[5] will not prohibit spontaneous hemostasis in splenic injury. However, spontaneous hemostasis of the lacerated enlarged spleen may be interfered with by the presence of portal hypertension that increases splenic intraparenchymal hydrostatic pressure. Besides, deficiency of coagulation factors with the resultant coagulopathy is common in patients with decompensated liver cirrhosis. With the concurrence of coagulopathy and portal hypertension, spontaneous hemostasis will be even more difficult. The feasibility of performing NOM in these patients should therefore be reevaluated.

The stratification of surgical risk in patients with liver cirrhosis can be performed in many ways. The preoperative Child's classification has been reported to be the best predictor of outcome before undertaking an elective operation.[10–14,18,19] In this study, Child's classification was difficult to assess. The presence and amount of ascites at the time of injury was impossible to measure because of associated intra-abdominal hemorrhage. No patient had encephalopathy before this injury. Serum bilirubin level was not significantly different between survivors and nonsurvivors. In this study, because of the limited number of patients, it is not statistically powerful enough to perform risk factor analysis leading to mortality in patients with concurrent liver cirrhosis and BSI. However, several factors show strong positive correlation with patient mortality. These include severely elevated PTs, high Injury Severity Scores, and decreased serum albumin levels. Because the ISS requires detailed survey and the serum albumin level is not checked emergently, PT seems to be the most readily available indicator before the development of hemodynamic instability.

Prothrombin time is the most critical variable for the high NOM failure rate and the subsequent mortality. In this study, except two patients without a preoperative PT, all 10 patients with available data had elevated PTs. Five patients with severely elevated PTs (more than two times the control values) died despite aggressive transfusion with blood and fresh frozen plasma (FFP) before, during, and after operation. As described by many other reports, coagulopathy is a risk factor for cirrhotic trauma victims.[11,12,18] If coagulopathy is induced or aggravated by the ongoing hemorrhage, early surgical intervention for hemostasis will be crucial. Any delay in surgical intervention would then induce unfavorable effects on the patient's outcome. In contrast, if coagulopathy is a preinjury comorbidity that causes ongoing hemorrhage, correction of the coagulopathy instead of surgical intervention will be imperative. In this study, without the knowledge of the patient's preinjury PTs, we are not able to determine whether the actual sequence is active splenic hemorrhage with subsequent development of coagulopathy or preexistent coagulopathy with continuous hemorrhage. However, according to our data, once severe coagulopathy is detected, it is very difficult to correct by supplying coagulation factors alone. Despite aggressive transfusion, all patients soon become hemodynamically unstable and require emergent laparotomy. Further attempt of NOM is obviously not suitable for them. These patients probably should not be treated nonoperatively from the beginning even if they present with stable hemodynamic status. More data are required before this recommendation can be made confidently because the patient population is quite small in this study.

In patients with mildly to moderately elevated PTs (less than two times the control values), controversies exist between early surgical intervention and aggressive correction of coagulopathy. Previous studies showed that abdominal operation in liver-cirrhotic patients was associated with a high mortality rate.[11–14] The mortality rate of emergency surgery in patients with advanced liver cirrhosis was reported to be as high as 86%.[19] Attempts to avoid emergent laparotomy by aggressive correction of coagulopathy may be beneficial to these patients. However, this treatment modality carries the risk of delaying operation, increasing the amount of transfusion, and inducing consumption coagulopathy and hepatic decompensation. In this study, five patients had mildly to moderately elevated PTs. Despite aggressive preoperative transfusion with FFP, four patients eventually required laparotomy and splenectomy. Active bleeding from the lacerated spleen was found during laparotomy in every patient. The possibility of achieving spontaneous hemostasis by more FFP transfusion is quite low. With ongoing hemorrhage and continuous blood transfusion, these patients may develop severe coagulopathy or encephalopathy and result in even worse outcome. Unless there are new therapies to correct coagulopathy, it would not be appropriate to insist on NOM for these patients. Lowering the threshold for operation and early termination of NOM for patients with coexistent BSI and liver cirrhosis may decrease the amount of transfusion and improve patient outcome.

We recommend checking PT as a primary survey for every trauma patient with a known history of liver cirrhosis. Emergent laparotomy along with aggressive transfusion of FFP should be performed in patients with severely elevated PT regardless of the hemodynamic status. Detailed assessment of the ISS and injury severity grade of the spleen will be reserved for patients with slightly to moderately elevated PT and stable hemodynamic status. High ISS or high-grade splenic injury (grade III–V) will favor emergent laparotomy.

For patients with low-grade splenic injury, low ISS, and normal to mildly elevated PT, nonoperative treatment will be carried out with great care. These patients should be admitted to the intensive care unit for close monitoring and aggressive maintenance of coagulation function. Once signs of continuous hemorrhage are detected, laparotomy should be arranged without delay.

REFERENCES

1. Bee TK, Croce MA, Miller PR, et al. Failure of splenic nonoperative management: is the glass half empty or half full? *J Trauma.* 2001; 50:230–236.
2. Myers JG, Dent DL, Stewart RM, et al. Blunt splenic injuries: dedicated trauma surgeons can achieve a high rate of nonoperative success in patients of all ages. *J Trauma.* 2000;48:801–806.
3. Cocanour CS, Moore FA, Ware DN, Marvin RG, Duke JH. Age should not be a consideration for nonoperative management of blunt splenic injury. *J Trauma.* 2000;48:606–612.
4. Barone JE, Burns G, Svehlak SA, et al. Management of blunt splenic trauma in patients older than 55 years. *J Trauma.* 1999; 46:87–90.
5. Pachter HL, Guth AA, Hofstetter SR, Spencer FC. Changing patterns in the management of splenic trauma: the impact of nonoperative management. *Ann Surg.* 1998;227:708–719.
6. Cocanour CS, Moore FA, Ware DN, et al. Delayed complications of nonoperative management of blunt adult splenic trauma. *Arch Surg.* 1998;133:619–625.
7. Velmahos GC, Chan LS, Kamel E, et al. Nonoperative management of splenic injuries: have we gone too far? *Arch Surg.* 2000;135:674–681.
8. Safary A, Beck J. Vaccination against hepatitis B: current challenges for Asian countries and future directions. *J Gastrohepatol.* 2000; 15:396–401.
9. Maddrey WC. Alcohol-induced liver disease. *Clin Liver Dis.* 2000; 4:115–131.
10. Maull KI, Turnage B. Trauma in the cirrhotic patient. *South Med J.* 2001;94:205–207.
11. Wahlstrom K, Ney AL, Jacobson S, et al. Trauma in cirrhotics: survival and hospital sequelae in patients requiring abdominal exploration. *Am Surg.* 2000;66:1071–1076.
12. Tinkoff G, Rhodes M, Diamond D, Lucke J. Cirrhosis in the trauma victim: effect on mortality rates. *Ann Surg.* 1990;211:172–177.
13. Rice HE, O'Keefe GE, Helton WS, Johansen K. Morbid prognostic features in patients with chronic liver failure undergoing nonhepatic surgery. *Arch Surg.* 1997;132:880–885.
14. Mansour A, Watson W, Shayani V, Pickleman J. Abdominal operations in patients with cirrhosis: still a major surgical challenge. *Surgery.* 1997;122:730–736.
15. Fang JF, Chen RJ, Lin BC, et al. Cell count ratio: new criterion of diagnostic peritoneal lavage for detection of hollow organ perforation. *J Trauma.* 1998;45:540–544.
16. Davis KA, Fabian TC, Croce MA, et al. Improved success in nonoperative management of blunt splenic injuries: embolization of splenic artery pseudoaneurysms. *J Trauma.* 1998;44:1008–1015.
17. Wasvary H, Howells G, Villalba M, et al. Nonoperative management of adult blunt splenic trauma: a 15-year experience. *Am Surg.* 1997; 63:694–699.
18. Morris JA, Mackenzie EJ, Edelstein SL. The effect of preexisting conditions on mortality in trauma patients. *JAMA.* 1990;263:1942–1946.
19. Arahna GV, Greenlee HB. Intra-abdominal surgery in patients with advanced cirrhosis. *Arch Surg.* 1986;121:275–277.

EDITORIAL COMMENT

Dr. Fang and colleagues have presented 12 patients with blunt splenic trauma and preexisting hepatic cirrhosis, with a mortality rate of 50%. They conclude that the prothrombin time (PT) should be checked in patients with splenic trauma and cirrhosis. They recommend prompt laparotomy for those with markedly elevated PTs and suggest that patients with mild PT elevations can safely be observed. It is hard to disagree with that conclusion, but there are a few issues with the article.

Two patients with injury-to-operation intervals of only 3 and 4 hours were included. Classifying these patients as having had a period of observation is problematic when one takes into account the amount of elapsed time for the prehospital care and time needed for resuscitation, examination, and computed tomographic scanning in hospital.

The amount of blood transfused ranged from 4 to 26 units, yet their protocol called for exploration of patients after 4 units were given. This discrepancy could be because the authors reported the amount of blood transfused in the first 72 hours. Mean time to the operating room was approximately 50 hours in both the cirrhotic and noncirrhotic patients. The article would have been more enlightening had they simply listed the amount of blood transfused in the observation period. Because Injury Severity Scores (ISSs) may not be accurately assessed until late in a patient's hospital course, caution should be exercised in attempting to relate the ISS to the likelihood of successful nonoperative management of a patient in real time.

Intuitively, one would suspect that cirrhotic patients would do poorly. This article does not actually prove that. What it documents is that cirrhotic patients with blunt splenic injury died if they had markedly elevated PTs. The article actually shows that marked elevation of PT (perhaps not necessarily cirrhosis) is associated with mortality. For the five deaths for which the PT was recorded, the mean was 85 $\pm$ 32. The five survivors with recorded PTs had a mean of 15.8 $\pm$ 1.8. Using the *t* test, $p = 0.001$.

In the last paragraph of the article, they state that in cirrhotic patients with "low-grade splenic injury, low ISS, and normal to mildly elevated PT, nonoperative treatment will be carried out with great care." I suggest that statement is true for all splenic trauma patients with or without cirrhosis. Patients with markedly elevated PTs should not undergo nonoperative management of blunt splenic injury regardless of the presence or absence of cirrhosis.

James E. Barone, MD, FACS, FCCM
Department of Surgery
The Stamford Hospital
190 West Broad Street
Stamford, CT 06902
drjbarone@stamhosp.chime.org