Cirrhosis in the Trauma Victim

Effect on Mortality Rates

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To evaluate the impact cirrhosis has on survival the records of 40 cirrhotic trauma victims from the registries of two Level 1 trauma centers were reviewed and probability of survival calculated using the TRISS methodology. Mechanism of injury, anatomic location, involvement of single or multiple sites, presence of ascites, elevations in serum glutamic oxaloacetic transaminase (SGOT), alkaline phosphatase, serum bilirubin, prothrombin time (PT), and hypoalbuminemia were tabulated for each patient. Contingency tables were created for injury and hepatic parameters, as related to survival, and subjected to chi square analysis. Loglinear analysis was performed on all significant parameters to evaluate the independent effects of injury characteristics and hepatic insufficiency on survival. Predicted survival was 93%; observed survival was 70% ($Z = -6.92$; $p < 0.001$). Cause of death was multiple-system organ failure (9) and closed head injury (3). Admission markers of poor outcome included one or more of the following: ascites, elevated PT or bilirubin, history of motor vehicle accident, multiple trauma, or blunt abdominal trauma requiring laparotomy. Loglinear analysis revealed that the presence of ascites, elevated PT, or bilirubin, further diminished the rate of survival for any individual injury characteristic. We concluded that survival among cirrhotic trauma victims was significantly lower than predicted. In addition the presence of hepatic insufficiency further diminishes survival, regardless of the injury sustained.

HEPATIC CIRRHOSIS IS the predominant cause of death in approximately 30,000 patients each year.[1] It is the fifth leading cause of death in individuals aged 45 to 64 years.[2] Cirrhosis, as a comorbid factor, also contributes to the demise of many more patients.

Many reports have shown that patients with cirrhosis who undergo elective or emergency surgery have significantly increased mortality and morbidity rates.[3–15] Also the degree of hepatic insufficiency is a prime factor in determining outcome in these patients.

Despite interest in the outcome of the cirrhotic surgical patient, there are, to date, no reports regarding the outcome of cirrhotic patients after traumatic injury. This study reviews the experience at two trauma centers with cirrhotic patients who sustained various injuries and characterizes the effect of this premorbid state on their outcome.

Materials and Methods

Data on more than 27,000 patients admitted to the trauma service at Lehigh Valley Hospital Center and Allegheny General Hospital over a 9-year period are contained in their respective trauma registries. These data were analyzed for patients filed under the ICDM-3 index for chronic liver disease with cirrhosis (571.0). The trauma registry abstract and medical record of all patients with this diagnosis were reviewed. Diagnosis of hepatic cirrhosis was confirmed by past medical history, clinical exam, operative findings, biopsy, and/or imaging. Patients were characterized by age, sex, trauma score, injury severity score, mechanism of injury, and outcome. These profiles were then analyzed for predicted outcome as compared to observed outcome. Predicted outcome was calculated for each individual and the group using the TRISS probability of survival.[16]

Patients' injuries were further characterized by anatomic location and involvement of single or multiple sites. In addition evidence of hepatic insufficiency was evaluated by assessing admission data for the presence of ascites, hyperbilirubinemia (more than 2 mg/dL), hypoalbumin-

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emia (less than 3.5 mg/dL), elevations in alkaline phosphatase (more than 125 μ/L), serum glutamic oxaloacetic transaminase (SGOT more than 40 μ/L), and/or prothrombin time (more than 12.5 seconds). For both the above injury profile and hepatic insufficiency profile, contingency tables were created for presence or absence of each parameter as related to outcome. These were subjected to chi square analysis with the significance level set at $p < 0.05$. Stepwise loglinear analysis using BMDP4F[17] was performed on all selected parameters to assess the combined and individual effect of specific injury characteristics and hepatic insufficiency on survival.

Results

Forty patients (1.4 per 1000 trauma admissions) admitted to the trauma service at the Lehigh Valley Hospital Center (24 patients) and Allegheny General Hospital (16 patients) from January 1979 to June 1988 had evidence of hepatic cirrhosis on clinical pathologic, and/or radiologic grounds. In 35 patients the diagnosis was made before admission during previous hospitalization, or as an outpatient. Twenty-two patients had diagnostic biopsy and 13 patients had the diagnosis made on clinical grounds, confirmed by radiologic and laboratory evidence. The remaining five patients were diagnosed during exploratory laparotomy for traumatic injury (two patients) or at autopsy (three patients). In 33 patients the etiology of the cirrhosis was related to alcohol. In six patients cirrhosis was cryptogenic, although in one patient Methyldopa toxicity was suggested as an inciting factor. The remaining patient had primary biliary cirrhosis.

The demographic and outcome data for the whole group are contained in Table 1. Noteworthy is that the predicted survival as reflected by TRISS was significantly greater ($p < 0.05$) than the observed survival (93% *vs.* 70%) with an associated Z statistic of −6.92 and M statistic of 0.86.

Although the predominant mechanism of injury was accidental fall (Table 2), 58% (7 of 12) of deaths were due to motor vehicle accidents. Other mechanisms of injury included a superficial abdominal stab wound, a criminal assault, and a 30% total-body-surface area burn.

TABLE 1. *Demographic and Survival Data*

(n = 40)*	Range
Mean age = 58	31–84
Mean TS = 15	6–16
Mean ISS = 14	5–34
TRISS = 0.93	
Surv = 0.70†	

* There were 28 men and 12 women.
† $p < 0.05$ as compared to TRISS.

TABLE 2. *Mechanism of Injury*

Mechanism of Injury	Total	Nonsurvivors (%)
Fall	25	4 (14)
MVA	12	7 (58)
Other	3	1 (33)

Table 3 lists the injuries by anatomic location. Fifteen patients had evidence of head trauma (closed head injury or skull fracture). Six of these patients died. In three of these six patients, head injury was the predominant cause of death.

Seven patients sustained blunt thoracic trauma including rib fractures, pulmonary contusion, or hemopneumothorax. One patient had both EKG and echocardiographic evidence of myocardial contusion. All patients in this group, including nonsurvivors, had significant associated injuries.

Blunt abdominal trauma evidenced by hemoperitoneum, splenic rupture, and/or liver laceration was the predominant injury in nine patients. Eight patients underwent laparotomy. The remaining patient, a known cirrhotic, had computed tomographic evidence of a class I liver laceration and was observed. Six of these patients, including the patient who was observed, died.

Pelvic fractures or extremity long-bone fractures occurred in 25 patients. Although eight deaths occurred in this group, five patients had more serious associated injuries. In fact, of the 12 who sustained pelvic fracture or extremity long-bone fracture as the single site of injury, only two patients died, succumbing to hepatic failure after lengthy hospitalizations. Of the remaining injuries, one patient sustained a superficial stab wound to the abdomen, another sustained serious maxillofacial trauma, and a third person died from a thermal injury.

Thirteen patients had injuries involving multiple sites. Nine of these patients died while hospitalized. The remaining 27 patients had injuries involving a single site; of these, three died.

The presence of clinical or laboratory findings associated with hepatic insufficiency in this group of patients

TABLE 3. *Injury Characteristics*

Site	Total	Nonsurvivors (%)
Head	15	6 (40)
Thorax	7	4 (57)
Abdomen	9	6 (67)*
Pelvis/Ext	25	8 (32)
Multiple	13	9 (69)*
Single	27	3 (11)

* $p < 0.05$.

TABLE 4. *Presence of Hepatic Insufficiency*

Parameters of Hepatic Insufficiency†	Total	Nonsurvivors (%)
Ascites	19	9 (47)*
SGOT > 40 μ/L	13	6 (46)
Alk phos. > 125 μ/L	13	6 (46)
Ser bili. > 2.0 mg/dL	17	10 (60)*
Ser alb. ≤ 3.0 mg/dL	22	7 (32)
PT > Control	15	8 (53)*

* $p < 0.05$.

† As determined by clinical exam and laboratory data on admission.

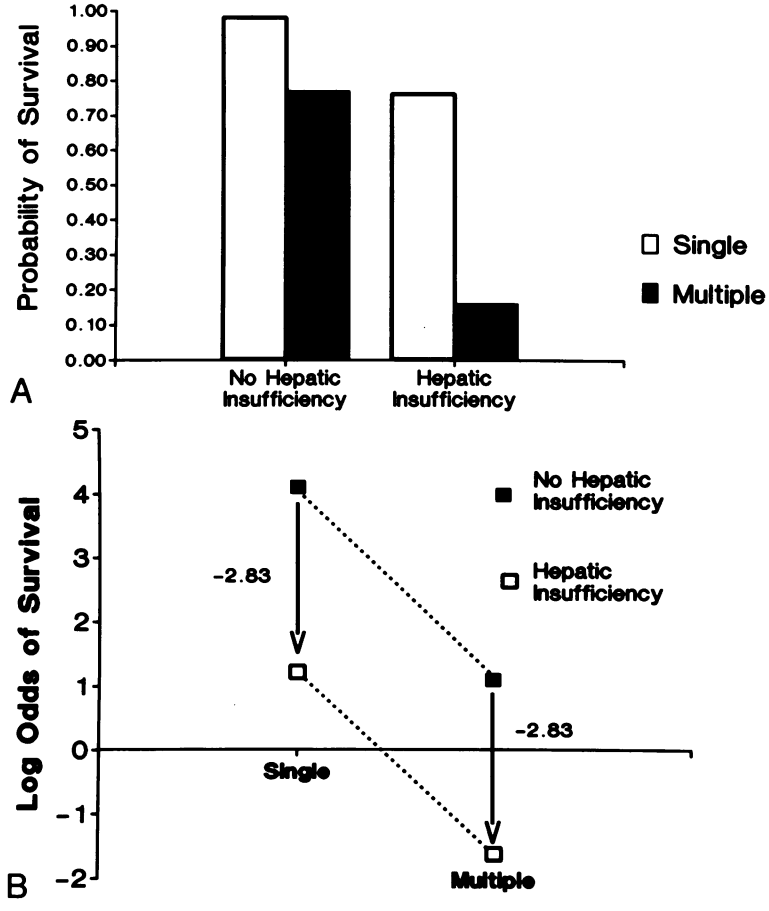
SGOT, serum glutamic oxaloacetic transaminase; Alk phos., alkaline phosphate; Ser bili., serum bilirubin; Ser alb., serum albumin; PT, alkaline phosphatase.

is outlined in Table 4. For comparison, the presence of these parameters in the 12 nonsurvivors is listed as well. The presence of ascites was confirmed by operative findings or radiologic evidence in 19 patients. Of these, nine died. Elevation of prothrombin time to more than 12.5 seconds was found in 15 patients, including eight nonsurvivors. Serum bilirubin in excess of 2 mg/dL was found in 17 patients on admission, of whom ten died (50%). The presence of any of these parameters was associated with significant increases ($p < 0.05$) in mortality. Elevations in SGOT and alkaline phosphatase and hypoalbuminemia were present in various patients, but did not significantly affect outcome.

Twenty-two patients had evidence of ascites, hyperbilirubinemia and/or elevated prothrombin time. This group's mortality rate was 50%, which was significantly greater ($p < 0.05$) than the predicted mortality rate of 12%. In the 18 patients without any abnormality in these parameters, only one death occurred, yielding a 6% mortality rate, which was not significantly different from predicted.

Loglinear analysis revealed that these parameters of hepatic insufficiency (ascites, hyperbilirubinemia, and elevated prothrombin time) and the presence of multiple injuries independently affected patient outcome. Both had a uniform negative effect on survival (Fig. 1).

Of the 12 patients who did not survive, three patients died of closed head injury; the remaining nine patients succumbed to multiple-system organ failure. In these nine patients, hepatic failure was either the inciting factor or played an integral role in the patient's demise. The demographics of this group are contained in Table 5.



FIGS. 1A and B. (A) Displays probability of survival of patients with single or multiple sites of injury as related to hepatic insufficiency. (B) Displays the uniform impact of hepatic insufficiency on log relative odds of survival when single or multiple sites of injury are involved.

Discussion

Hepatic cirrhosis invariably follows hepatocellular necrosis.[18] Cirrhosis, regardless of etiology, inhibits the liver's response to injury. The predominant histologic features are wide-spread fibrosis and nodule formation with loss of normal hepatic architecture. These changes are manifested clinically by hepatic failure and portal hypertension, the magnitude of which determines an individual patient's course and prognosis.

Adequate hepatic function is necessary in the physiologic response to surgery or traumatic injury. The liver performs a vital role in protein synthesis, detoxification, and immunologic response. In a patient subjected to surgical intervention or traumatic injury, any degree of hepatic insufficiency would diminish the liver's ability to carry out these vital metabolic functions. Thus, because of impaired cirrhotic reserve, a cirrhotic surgical or trauma patient would be at great risk for morbid complication or death during the recovery period.

The increased risk to the cirrhotic patient undergoing surgery has been well documented.[3–15,19–20] Essentially the basis of Child's criteria was to classify operative risk in cirrhotic patients undergoing portosystemic shunt surgery.[19] Child[20,21] showed that the degree of hepatic decompensation correlated with the rate of operative mortality in these patients. Furthermore other studies had shown that if therapeutic measures were taken to improve the cirrhotic patient's clinical status before operation and upgrade his Child's class, postoperative outcome after portosystemic shunting was improved.

In fact surgery of various types carries increased risk in the cirrhotic patient. In 1963 Lindenmuth and Eisenberg[15] studied 91 patients with varying degrees of cirrhosis. They reported an overall complication rate of 25% and a mortality rate of 8% after operative procedures, which ranged in magnitude from herniorrhaphy to major gastrointestinal surgery and included portosystemic shunting. A more recent review by Doeberneck et al.[5] in 1983 reported operative mortality rate as 19.6% and morbidity rate of the survivors as 47%. Interestingly 16 of the 20 patients who died underwent exploratory laparotomy and intra-abdominal surgery without portosystemic shunting. The mortality rate for this group alone was 35% while the operative mortality rate for the remainder of the patients undergoing herniorrhaphy, orthopedic repair, genitourinary surgery, or portosystemic shunting was 9%.

It has been appreciated that surgery not involving portosystemic shunting, but requiring laparotomy, poses a grave risk to the cirrhotic patient. Schwartz,[7] Cryer et al.,[8] and Arahna et al.[11] have studied cirrhotic patients undergoing biliary tract surgery. They reported mortality rates of 27%, 21%, and 24%, respectively. These rates are far in excess of the 1.7% reported morality rate for the noncirrhotic patient undergoing biliary tract surgery.[21] Furthermore Garrison and coworkers reviewed 100 consecutive patients with biopsy-proved cirrhosis undergoing various intra-abdominal operations.[6] Similar to the reviews previously cited,[7,8,11] the patients in this group who underwent surgery on their biliary tract had a mortality rate of 21%. In addition mortality rates for gastroduodenal surgery (35%), small bowel surgery (60%), colon resection (56%), and even open liver biopsy (20%), were exceedingly high as compared to rates in noncirrhotic patients. In 1986 Arahna et al.[4] reviewed their own experience with cirrhotic patients undergoing intra-abdominal surgery, exclusive of that involving the biliary tract or portosystemic shunting, and reported an overall operative mortality rate of 64%.

Despite these dismal figures, it is clear that certain cirrhotic patients undergo surgical intervention with only a moderate increase in risk. As alluded to before, those patients undergoing nonabdominal surgery fared reasonably well.[5,12,14] Furthermore, in all studies cited,[3–15] those pa-

TABLE 5. *Demographic Data on Nonsurvivors*

Age	Sex	TS	ISS	Mechanism	HD	Injury	OR	Cirr. Etiol.	Cause of Death
67	M	6	34	Fall	20	Multiple	Lap	ETOH	CHI, Sepsis
66	M	8	34	MVA	2	Multiple	Lap	ETOH	Coag, Sepsis, MSOF
68	M	14	20	MVA	7	Multiple	ORIF	ETOH	Hepatic failure, MSOF
53	M	15	26	MVA	11	Multiple	Lap	ETOH	Hepatic failure, MSOF
70	F	16	34	Explosion	6	Single	—	ETOH	Hepatic failure, MSOF
50	M	9	34	MVA	2	Multiple	ORIF	ETOH	CHI
76	F	16	9	Fall	30	Single	—	Crypt.	Hepatic failure, MSOF
46	M	4	34	MVA	7	Multiple	Lap	ETOH	Coag, Sepsis, MSOF
41	M	12	16	Fall	10	Single	—	ETOH	CHI, Pneumonia
64	M	14	14	Fall	25	Multiple	ORIF	ETOH	Hepatic failure, MSOF
72	F	16	14	MVA/PED	18	Multiple	ORIF	Crypt.	Hepatic failure, MSOF
57	F	15	22	MVA/PED	22	Multiple	Lap	ETOH	Hepatic failure, MSOF

HD, hospital days; Lap, laparotomy; ORIF, open reduction/internal fixation; CHI, closed head injury; Coag, Coagulopathy; MSOF, multiple system organ failure; MVA, motor vehicle accident; PED, pedestrian-motor vehicle accident; ETOH, ethanol; crypt, cryptogenic.

tients with only minimal evidence of impaired hepatic function had significantly lower morbidity and mortality rates than patients who had clinical and laboratory findings of hepatocellular failure (*i.e.*, ascites, encephalopathy, elevated serum bilirubin, elevated prothrombin times, and/or decreased serum albumin). In fact Sirineck and colleagues[3] have recently shown that the mortality rate can be significantly reduced by instituting perioperative therapeutic measures (previously used before portosystemic shunting) to control ascites, correct abnormal coagulation defects, and nutritionally replete the cirrhotic surgical patient.

Most important, the cirrhotic patient does not tolerate emergency surgery. When compared to elective procedures, cirrhotic patients of similar Child's class had as much as a threefold increase in mortality rate.[4,6] Arahna et al.[4] reported a mortality rate of 86% in patients with advanced cirrhosis who underwent emergency surgery. This is understandable because no effort can be made to improve the clinical status in a cirrhotic patient undergoing emergency surgery. The cirrhotic trauma victim characteristically resembles the cirrhotic undergoing emergency surgery. Like emergency surgery, traumatic injury and its treatment allows little time for optimizing the cirrhotic patient's hepatic functions and clinical condition. Therefore it is apparent that the cirrhotic trauma patient is at grave risk and has an overall poor prognosis as does the cirrhotic undergoing emergency surgery. Interestingly we could find no published reports pertaining to cirrhosis in trauma victims. Garrison's[6] series included four cirrhotic patients who underwent exploratory laparotomy for trauma, three of whom died, but did not deal with these cases specifically. The intention of our study was to clarify this situation and characterize this group of patients.

As with the cirrhotic surgical patient, the mortality rate among our patients was higher than expected. The 30% mortality rate was significantly higher than predicted in relation to the severity of the specific injuries as reflected by TRISS. The associated Z statistic of -6.92 quantitates this difference. On the other hand, the M("match") statistic quantifies the difference in the distribution of probability of survival (P_s) ranges in our study population and the Multiple Trauma Outcome Study (MTOS) norm. The M statistic of 0.86 showed that our patients were distributed over higher P_s ranges, indicating that as a group they were less severely injured than the MTOS population.

Several factors portended poor outcome in our group. Although the predominant mechanism of injury was falls, those patients involved in motor vehicle accidents (MVA) fared the worst. Because the severity of associated injuries was increased with MVAs as the mechanism of injury, this outcome would be expected.

Regarding site of injury, patients sustaining blunt abdominal trauma with hemoperitoneum or visceral injury, requiring laparotomy, incurred a significant increase in mortality (67%). This mirrored the surgical cirrhotic patient in which the highest mortality rates were associated with laparotomy and intra-abdominal surgery. The presence of multiple injuries increased total injury severity and, as expected, was the injury characteristic associated with the highest increase in mortality rate (69%). On the other hand there were only three deaths (11%) in the group of 27 patients with injuries involving a single site.

Child's criteria could not be used to classify these patients. Nutritional status and response to therapy were unobtainable. Furthermore mental status changes on admission could not be solely attributed to hepatic etiology. However the evidence of hepatic insufficiency, as determined by the presence of ascites and/or elevated prothrombin time, correlated with outcome in these patients, as it has in patients undergoing operative intervention. Multivariate analysis revealed that the cirrhotic trauma victim presenting with ascites, hyperbilirubinemia, or elevation in prothrombin time exhibited a uniformly lower rate of survival independent of injury characteristics. In fact, of the seven patients presenting with all three abnormalities, none survived.

Certainly a study population of this size limits the information that can be yielded. A multicenter study, using urban populations, will be necessary to fully evaluate and characterize the effects of cirrhosis on patients sustaining traumatic injuries. Until such information can be compiled, the data embodied in this review confirms the similarity of the cirrhotic trauma patient to the cirrhotic patient undergoing emergency surgery. As with surgery patients, cirrhosis in the trauma victim is associated with a significant increase in mortality rate. Admission markers of poor outcome include one or more of the following: ascites, hyperbilirubinemia, elevated prothrombin time, multiple injuries, or blunt abdominal trauma requiring laparotomy. Finally the presence of hepatic insufficiency on admission increases mortality rate regardless of the injury sustained.

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