

Evaluation of liver injury in a tertiary hospital: a retrospective study

Ismail Bilgiç, M.D.,¹ Sibel Gelecek, M.D.,¹ Ali Emre Akgün, M.D.,¹ Mehmet Mahir Özmen, M.D.²

¹Department of General Surgery, Ankara Numune Training and Research Hospital, Ankara;

²Department of General Surgery, Hacettepe University Faculty of Medicine, Ankara

ABSTRACT

BACKGROUND: Liver is the most frequently injured intraabdominal organ following abdominal trauma. Liver injury in polytraumatized patients can vary from minor contusions to major lacerations and is associated with morbidity and mortality. The objective of this study was to evaluate the outcome of liver injury in polytraumatized patients.

METHODS: Only surgically treated 82 patients with liver injury over an eight year period (2005-2013) were included in this study and analyzed retrospectively. Data collected included demographics, laboratory findings, intraoperative findings, operative management, and outcome. The patients were divided into two groups and the mortality and survival data were compared.

RESULTS: The overall mortality rate was 18.3% (15 of 82 patients). 34 (41.5%) patients had blunt, forty-eight (48.5%) had penetrating trauma. There were multiple traumas in forty-seven (57%) patients. Forty-seven (57%) patients had total of seventy one coexisting intraabdominal injuries. Forty-six (56.1%) patients had stable and thirty-six (43.9%) had unstable hemodynamics on admission. In mortality group AST, ALT, LDH, APTT, PT, INR, and creatinine levels were high, fibrinogen levels and platelet counts were low on admission.

CONCLUSION: Hemodynamic instability, coexisting musculoskeletal and chest injury, high APTT, PT, INR, AST, ALT, LDH levels, and low fibrinogen levels and platelet counts on admission should be considered as predictive factors for mortality.

Key words: Liver enzymes; liver injury; mortality.

INTRODUCTION

Liver is the most frequently injured organ following abdominal trauma^[1] and associated injuries contribute significantly to morbidity and mortality. Liver is a particularly vulnerable organ because of its size and the fixed position in the right hypochondria. Mortality rates have fallen from 66% in World War II to current levels of 28%;^[2,3] however, mortality rates from complex liver trauma still remain high despite improvements in resuscitation, anesthesia and intensive care facilities.^[4]

The aim of this retrospective study was to document the outcome of the patients treated operatively.

MATERIALS AND METHODS

This retrospective clinical study was performed in the Emergency Service of Ankara Numune Teaching and Research Hospital between August 2005 and January 2013. Only surgically treated 82 patients were included in this study and analyzed. All patients had either unstable hemodynamics or signs of abdominal injuries requiring operation. Clinical data regarding patient demographics, AST (aspartate aminotransferase), ALT (alanine aminotransferase), LDH (lactate dehydrogenase), APTT (activated partial thromboplastin time), PT (prothrombin time), INR (international normalized ratio), fibrinogen, urea, creatinine, hemoglobin levels, platelet counts, and white blood cell counts on admission, mechanism of injury, hemodynamic status on presentation, hepatic injury grade, operative procedures, coexisting abdominal and extra-abdominal injuries, number of blood transfusion, outcome in terms of liver injury related morbidity and mortality were collected. Coexisting extra-abdominal injuries were divided broadly into cranial injury, chest injury (including rib fracture, haemothorax or pneumothorax, lung and cardiac), musculoskeletal system injuries (including long bone fracture) and retroperitoneal hematoma (including pelvic fracture and great vessel injury).

Address for correspondence: İsmail Bilgiç, M.D.

Ülkü Mah., Talatpaşa Bulvarı, No: 5, Altındağ, 06100 Ankara, Turkey

Tel: +90 312 - 508 51 33 E-mail: drismailbilgic@gmail.com

Quick Response Code



Ulus Travma Acil Cerrahi Derg
2014;20(5):359-365
doi: 10.5505/tjtes.2014.22074

Copyright 2014
TJTES

In accordance with the current Advanced Trauma Life Support (ATLS) protocols, patients were divided into two main groups according to the hemodynamics at presentation: stable and unstable.^[5] Hemodynamic instability was defined as systolic blood pressure less than 90 mmHg and pulse rate higher than 100 beats per minute at the time of presentation in our hospital. Three patients were in cardiopulmonary arrest on admission and were operated soon after resuscitation. Those patients were classified in a third group called “cardiopulmonary arrest”.

Based on the operation record, liver injuries were graded according to the Organ Injury Scale of the American Association for the Surgery of Trauma.^[6] The surgical techniques used to control bleeding during the operations were grouped into five categories including non-therapeutic, simple hemostatic measures, hepatorrhaphy with primary saturation, segmentectomy and lobectomy. Intervention was considered non-therapeutic when no active bleeding from the liver injury was detected. Superficial lacerations were managed by simple hemostatic measures such as diathermy and application of biomaterials (collagen sponge, oxidized regenerated cellulose gauze). Hepatorrhaphy was performed applying deep horizontal mattress stitches with polyglycolic acid. Peripherally located large lacerated segments were resected in non-anatomical fashion. Lobectomy was carried out as right hepatectomy in anatomical fashion.

Statistical Method

Data were analyzed using the statistical package, PASW 18.0 for Windows (SPSS, Chicago, IL, USA). The Kolmogorov-Smirnov test was applied to evaluate the distribution of values in continuous variables. The chi-squared test or Fischer's exact test was used to compare categorical data. For the parametric distribution, Student's t-test was used to compare the mean values of two groups. For nonparametric variables, the Kruskal-Wallis test or Mann-Whitney U-test was used to compare the median values of the response variable. Correlation analysis was performed using Pearson or Spearman. A stepwise logistic regression analysis was also carried out. The model included explanatory or predictive factors as variables suggested in the univariate analysis to be more strongly and significantly associated with mortality. Mortality was the outcome variable for multivariate analysis. The odds ratio (OR) was calculated as an estimate of relative risk between two groups on the basis of mortality as an outcome. Statistical significance was defined as $p < 0.05$.

RESULTS

From August 2005 to January 2013, 82 patients with liver trauma were surgically treated in the Department of Emergency Service of Ankara Numune Teaching and Research Hospital. Among these eighty-two patients, there were 77 (94%) males and 5 (6%) females, with a mean age of 34 years (range, 17-

90) (Table 1). The overall mortality rate was 18.3%. Patients' white blood cell count (WBC), platelet count, hemoglobin, AST, ALT, LDH, APTT, PT, INR, fibrinogen, urea and creatinine levels were measured on admission (Table 1). Age had no significant effect on mortality ($p = .08$); however, the female gender was found to be significant on mortality ($p = .04$).

Injury was defined as blunt trauma in thirty-four (41.5%) patients, stab wounds in thirty-eight (46.3%) and gunshot injury in ten (12.2%) patients. Forty-six (56.1%) patients had stable, thirty-three (40.2%) had unstable hemodynamics and three (3.7%) were in cardiopulmonary arrest on admission. Considering the intraabdominal injury; 35 patients had isolated liver injury and the remaining forty-seven (57%) had a total of seventy one coexisting intraabdominal injuries. Liver injury occurred as a component of multiple traumas in forty-seven (57%) patients. Musculoskeletal system injuries were the most common coexisting injuries (Table 2).

In operative findings, according to the Organ Injury Scale of the American Association for the Surgery of Trauma, 27 (33%) patients had Grade I, thirty-seven (45%) had Grade II, fifteen (18%) had Grade III, two (3%) had Grade IV and one (1%) had Grade V injuries (Table 2). No active bleeding from the liver in 10 (12%) patients undergoing non-therapeutic intervention during surgery was detected. Simple hemostatic measures such as diathermy, application of biomaterials (collagen sponge, oxidized regenerated cellulose gauze) were performed in fifteen (18%) patients. Hepatorrhaphy was the most common surgical procedure employed to control the bleeding in fifty-four (66%) patients. Two patients (3%) had peripherally located large lacerated segments and non-anatomical segmental resections were performed. A patient had central crush

Table 1. Laboratory findings

Parameter	Mean±SD
Age (year)	34.0 ±13.4
Hemoglobin (gr/dl)	13.1 ±2.3
White blood cells	14784.4±6940.3
Platelet (/mm ³)	246500±78200
Urea levels	34.07±19.7
Creatinine levels	1.08±.44
Aspartate aminotransferase	277.6±382.4
Alanine aminotransferase	272.9±394.02
Lactate dehydrogenase	648.4±624.4
Activated partial thromboplastin time	33.2±3.7
Prothrombin time	16.4±1.0
International normalized ratio	1.3±0.8
Fibrinogen	195.6±89.5
Transfusion requirement	2.3±3.8 (0-16)

Table 2. Clinical features

	n	%
Type of injury		
Blunt trauma	34	41.5
Stab wounds	38	46.3
Gunshot wounds	10	12.2
Hemodynamic status on admission		
Cardiopulmonary arrest	3	3.7
Unstable	33	40.2
Stable	46	56.1
Grade of liver injury		
Grade I	27	33
Grade II	37	45
Grade III	15	19
Grade IV	2	2
Grade V	1	1
Operative procedure		
Non-therapeutic	10	12
Simple hemostatic measures	15	18
Hepatorrhaphy	54	66
Segmentectomy	2	3
Lobectomy	1	1
Coexisting intraabdominal injury		
Diaphragm	18	25
Spleen	13	18
Stomach	11	16
Kidney	8	11
Colon	7	10
Small intestine	5	7
Duodenum	4	6
Gallbladder	4	6
Pancreas	1	1
Coexisting extra-abdominal injury		
Musculoskeletal system	24	35
Chest trauma	22	32
Retroperitoneal injury	16	23
Cranial injury	7	10

injury resulting in a stellate-type laceration including segment 5, 6, 7, 8 and right hepatic vein injury in the liver. Right hepatectomy was performed for this patient (Table 2).

High levels of AST, ALT, LDH, APTT, PT, INR, creatinine and low levels of fibrinogen and low platelet counts on admission were found to be associated with mortality (Table 3). In addition, AST, ALT, APTT, PT, INR, and LDH levels on admission were found to be correlated with the grade of liver

injury (Table 4). LDH levels on admission were found as an independent risk factor for mortality in multivariate analysis ($p=0.008$). Preoperative blood transfusion requirement ranged from 0-16 units (mean: 2.3 ± 3.8) and it was statistically correlated with the grade of liver injury ($p=0.003$) (Table 4).

Hemodynamic instability on admission and the type of injury were found to be significantly correlated with mortality ($p=.001$ and $p=.04$, respectively) (Table 5).

Accompanying abdominal injuries were not observed to be associated with mortality; however, the grade of the liver injury, presence of musculoskeletal system and chest injury were found to be associated with mortality (Table 5).

Packing was performed in 6 patients. In those patients, three had grade III and two had grade IV and one had grade V injuries. Among the two patients who died in the packing group, one had grade IV and the other had grade III injuries. A significant relationship between the grade of the liver injury and packing application was observed (Table 4).

Out of 82 patients, fifteen did not survive. Two of them had isolated liver injury and both injuries were caused by blunt trauma. The mean age in the mortality group was 35 (range, 20-61) years. Among the fifteen patients, 12 (80%) were males and 3 (20%) were females. Ten (66.7%) patients had blunt trauma, 3 (20%) had stab wounds and 2 (13.3%) had gunshot wounds. One (6.7%) patient was in cardiopulmonary arrest, 2 (13.3%) were hemodynamically stable and 12 (80%) were hemodynamically unstable on admission. The grade of the liver injury, applied surgical technique and laboratory results were shown on Table 6.

Eight patients (9.75%) required re-laparotomy for various reasons including the removal of packs (3), no improvement (2), and intra-abdominal abscess formation (2). There was only one bile leak treated non-operatively.

DISCUSSION

Although splenic injuries are more common following blunt abdominal trauma, liver is the most frequently injured intra-abdominal organ.^[4] Complex liver injuries are still a challenging problem with high mortality rates (50%) despite improvements in resuscitation, surgical skills, anesthesia and intensive care. High grade liver injuries are usually associated with extra and intra-abdominal injuries due to high magnitude of the trauma, increasing the rate of mortality. The aim of this retrospective research was to evaluate the outcomes of surgically treated patients.

Seventy seven (94%) patients were male in our study. A male predominance has been demonstrated in almost every other liver trauma series, including studies from England (79%),^[7] other parts of Western Europe (67%- 74%),^[8-10] South Africa

Table 3. Significant laboratory findings for mortality

	Mortality group	Survival group	p
Age	34.8 (±11.5)	33.8 (±13.8)	.08
Hemoglobin	12.2 (±2.8)	13.3 (±2.1)	.117
White blood cells	18046 (±8687)	14111 (±6400)	.062
Platelet	205000 (±76000)	257000 (±75000)	.025
Urea	40.54 (±41.75)	32.65 (±10.16)	.193
Creatinine	1.4 (±.75)	1.0 (±.3)	.003
Aspartate aminotransferase	559.2 (±462.2)	217.3 (±338.2)	.001
Alanine aminotransferase	546.0 (±495.6)	215.4 (±347.7)	.001
Lactate dehydrogenase	1378 (±775.4)	533.2 (±521.5)	.009
Activated partial thromboplastin time	40.2 (23.8-133)	23.6 (17.5-51.9)	.000
Prothrombin time	20 (12.7-47)	13.8 (12-19)	.004
International normalized ratio	1.56 (1.02-3.75)	1.1 (.95-1.68)	.004
Fibrinogen	129.2 (±56)	219.7 (±85.77)	.002

Table 4. Factors correlated with the grade of the liver injury

	Grade of the liver injury	
	r	p
Aspartate aminotransferase	.346	0.004
Alanine aminotransferase	.324	0.007
Lactate dehydrogenase	.561	<0.001
Activated partial thromboplastin time	.30	0.046
Prothrombin time	.36	0.019
International normalized ratio	.36	0.02
Blood transfusion requirement	.322	0.003
Packing	.352	0.001

(81%)^[11] and North America (61%- 79%).^[12] Although the female gender was found to have a statistically significant effect on mortality, no homogeneity in gender distribution could be observed in our study as there were only five female patients. A higher frequency of liver trauma among younger patients was determined in the present study. Wilson et al.^[13] have reviewed many series of liver trauma and emphasized that the patient average age tended to lie between 25 and 30. Continuity of this global pattern has been shown in more recent works. Large liver trauma series have shown mean ages of 35, 32, and 30, respectively in Germany,^[10] South Africa,^[11] and North America.^[12]

In the present study, liver injuries were caused by blunt trauma in 41.5% of the patients (12.2% falls from height, 29.3% road traffic accidents) and penetrating trauma was seen in 58.5% of the patients (46.3% stab wounds, 12.2% gunshot

wounds). Scollay et al.^[14] have shown that European authors have reported high frequencies of blunt trauma. On the contrary, Krige,^[11] reporting the South African experience, found that 66% of 446 patients had sustained penetrating liver injury. Feliciano et al.^[15] have reviewed 1000 patients with liver trauma and found that penetrating trauma was responsible for 86% of hepatic injuries. On the other hand, incidence of stab wounds in penetrating trauma in the present study was similar to the European series having significantly higher incidence of stab injuries and a lower incidence of gunshot wounds.^[14] It has also been shown that blunt injuries have consistently been associated with a higher mortality rate than penetrating injuries.^[16,17] In the present study, mortality has been found to be significantly higher in blunt trauma patients. This might partly be explained by blunt traumas being usually associated with extra and intra-abdominal injuries increasing mortality rates due to the high magnitude of trauma.

Table 5. Gender, coexisting extra-abdominal and intra-abdominal injuries and hemodynamic status

	Mortality group		Survivor group		p
	n	%	n	%	
Female (Gender)	3	20	2	3	.04
Type of trauma (Blunt/penetrant)	10/5	29/10	24/43	71/90	.04
Musculoskeletal system	8	33.3	16	66.7	.03
Chest trauma	8	36.4	14	63.6	.02
Retroperitoneal injury	4	25	12	75	.5
Cranial injury	3	42.9	4	57.1	.1
Diaphragm	4	22.2	14	77.8	.7
Spleen	4	30.8	9	69.2	.2
Stomach	2	18.2	9	81.8	1.0
Kidney	1	12.5	7	87.5	1.0
Colon	3	42.9	4	57.1	.1
Hemodynamic instability	12	48	21	31	.001

In the present study, hemodynamic instability was recorded in 40% of liver trauma patients. These patients had a higher mortality rate than those who had a blood pressure higher than 90 mmHg ($p=0.001$). Several studies have identified hemodynamic instability as an early predictor of outcome after severe injuries.^[4,9,14,18-21] It has been shown by Clarke et al.^[22] that mortality increases by 1% every 3 minutes after a trauma involving hemotogenic shock. Also, Wilson have emphasized that shock on admission is thought to double the mortality rates.^[23] High mortality rates due to hemodynamic instability may be the result of severe exsanguinating hemorrhage resulting in catastrophic final pathway of hypothermia, coagulation, and acidosis.

Table 6. Demographic and clinical features of the mortality group

	n	%
Grade of liver injury		
Grade I	1	6.7
Grade II	6	40
Grade III	6	40
Grade IV	1	6.7
Grade V	1	6.7
Operative procedure		
Non-therapeutic	3	20
Simple hemostatic measures	1	6.7
Hepatorrhaphy	9	60
Segmentectomy	1	6.7
Lobectomy	1	6.7

Hemorrhage accounts for over the third of early trauma deaths^[24] and is a leading cause of preventable mortality.^[25] Acute traumatic coagulopathy is known to occur in about 28% to 34% of patients with multiple injuries.^[26] Most of the literature characterizes the condition by reference to an elevated INR, PT and/or PTT of 1.5 or 2 times normal.^[27] It has been shown in many studies that patients arriving in the emergency department with a coagulopathy are three to four times more likely to die and eight times more likely to die within the first 24 hours.^[26,28,29] In the present study, we found that PT, APTT and INR were significantly high in the mortality group than in the survivors. There are controversies about fibrinogen levels in acute traumatic coagulopathy. Martini et al.^[30] have shown that fibrinogen levels rarely decrease in patients with acute traumatic coagulopathy. On the other hand, it has been shown that fibrinogen concentrations rapidly decline after injury especially in hypoperfusion.^[31] Significantly low fibrinogen levels were detected in the mortality group. Platelet counts are mildly reduced by trauma and this appears to be associated with poor outcomes.^[31] Brown et al.^[32] have reported that platelet count on admission is inversely correlated with 24-hour mortality. In the present study, low platelet counts were significantly correlated with mortality. All these parameters are important since recent therapeutic and observational studies have demonstrated improved survival rates with better and early management of hemostasis after injury.^[31] Additionally, early identification of the need for massive transfusion (MT) may increase the speed and success of hemostatic intervention in trauma patients. The use of massive transfusion protocols standardizing blood component therapy that automatically delivers at specific points within resuscitation may be of benefit to prevent and treat early coagulopathy.

It is well-known that liver injuries almost always accompany injuries to other organ systems and liver injury has been found to be associated with high mortality rates with the presence of coexisting injuries.^[33,34] Also, Nishida et al.^[18] have founded that the presence of a coexisting injury is an independent prognostic factor for mortality in their multivariate analysis. In the present study, the presence of musculoskeletal injury and chest injury were both found to be associated with high mortality rates ($p=.03$ and $p=.02$).

Elevated serum liver enzymes, AST and ALT, are known to be associated with blunt traumatic liver injury. It has been shown in animal models and human studies that not only does the increase in the enzyme occur within a few hours after blunt liver trauma, but the amount of the increase in the enzyme also correlates to the severity of liver injury, as in the present study.^[35] Similarly, statistically significant and increasing ALT levels were observed among patients with increasing grades of liver injury.^[7] Tan et al.^[21] have reported that there is an important relationship between ALT, AST and hepatic injuries after blunt abdominal trauma and also patients with normal ALT, AST and LDH are unlikely to have major liver injury. Nishida et al.^[18] have shown that ALT is an independent risk factor for mortality in their multivariate analysis. AST and ALT levels on admission were found to be correlated with mortality and severity of liver injury in the present study.

LDH is a cytoplasmic enzyme present essentially in all major organ systems. The extracellular appearance of LDH is used to detect cell damage or cell death. It is released into the peripheral blood after cell death caused by ischemia, excess heat or cold, starvation, dehydration, injury, exposure to bacterial toxins, ingestion of certain drugs, and chemical poisonings. Due to its extraordinarily widespread distribution in the body, the total serum LDH is a highly sensitive, but nonspecific test. In the present study, LDH levels on admission were found to be correlated with mortality and severity of the liver injury; in addition, LDH was found as an independent risk factor for mortality in multivariate analysis in the present study. High LDH levels may reflect the number and severity of affected organs. AST, ALT and LDH together may be useful for prognostic factors in liver injury.

Conclusion

Hemodynamic instability, coexisting musculoskeletal and chest injury, and high APTT, PT, INR, AST, ALT, LDH levels and low fibrinogen levels and low platelet counts on admission should be considered as predictive factors for mortality in patients who sustained liver injury due to trauma. It is crucial to keep in mind that the high AST, ALT and LDH levels on admission might also reflect the high grade of liver injury.

Conflict of interest: None declared.

REFERENCES

1. Feliciano DV. Surgery for liver trauma. *Surg Clin North Am* 1989;69:273-84.
2. Richardson JD. Changes in the management of injuries to the liver and spleen. *J Am Coll Surg* 2005;200:648-69. [CrossRef](#)
3. Trunkey DD. Hepatic trauma: contemporary management. *Surg Clin North Am* 2004;84:437-50. [CrossRef](#)
4. Parks RW, Chryso E, Diamond T. Management of liver trauma. *Br J Surg* 1999;86:1121-35. [CrossRef](#)
5. American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support Student Manual*. 5th ed. Chicago, Illinois: American College of Surgeons; 1995.
6. Moore EE, Cogbill TH, Jurkovich GJ, Shackford SR, Malangoni MA, Champion HR. Organ injury scaling: spleen and liver (1994 revision). *J Trauma* 1995;38:323-4. [CrossRef](#)
7. John TG, Greig JD, Johnstone AJ, Garden OJ. Liver trauma: a 10-year experience. *Br J Surg* 1992;79:1352-6. [CrossRef](#)
8. Talving P, Beckman M, Häggmark T, Iselius L. Epidemiology of liver injuries. *Scand J Surg* 2003;92:192-4.
9. Menegaux F, Langlois P, Chigor JP. Severe blunt trauma of the liver: study of mortality factors. *J Trauma* 1993;35:865-9. [CrossRef](#)
10. Matthes G, Stengel D, Seifert J, Rademacher G, Mutze S, Ekkernkamp A. Blunt liver injuries in polytrauma: results from a cohort study with the regular use of whole-body helical computed tomography. *World J Surg* 2003;27:1124-30. [CrossRef](#)
11. Krige JE, Bornman PC, Terblanche J. Liver trauma in 446 patients. *S Afr J Surg* 1997;35:10-5.
12. Pachtel HL, Knudson MM, Esrig B, Ross S, Hoyt D, Cogbill T, et al. Status of nonoperative management of blunt hepatic injuries in 1995: a multicenter experience with 404 patients. *J Trauma* 1996;40:31-8. [CrossRef](#)
13. Wilson RH, Moorehead RJ. Hepatic trauma and its management. *Injury* 1991;22:439-45. [CrossRef](#)
14. Scollay JM, Beard D, Smith R, McKeown D, Garden OJ, Parks R. Eleven years of liver trauma: the Scottish experience. *World J Surg* 2005;29:744-9. [CrossRef](#)
15. Feliciano DV, Mattox KL, Jordan GL Jr, Burch JM, Bitondo CG, Cruse PA. Management of 1000 consecutive cases of hepatic trauma (1979-1984). *Ann Surg* 1986;204:438-45. [CrossRef](#)
16. Fabian TC, Croce MA, Stanford GG, Payne LW, Mangiante EC, Voeller GR, et al. Factors affecting morbidity following hepatic trauma. A prospective analysis of 482 injuries. *Ann Surg* 1991;213:540-8. [CrossRef](#)
17. Rivkind AI, Siegel JH, Dunham CM. Patterns of organ injury in blunt hepatic trauma and their significance for management and outcome. *J Trauma* 1989;29:1398-415. [CrossRef](#)
18. Nishida T, Fujita N, Nakao K. A multivariate analysis of the prognostic factors in severe liver trauma. *Surg Today* 1996;26:389-94. [CrossRef](#)
19. Sikhondze WL, Madiba TE, Naidoo NM, Muckart DJ. Predictors of outcome in patients requiring surgery for liver trauma. *Injury* 2007;38:65-70.
20. Velasco RA, Martínez FB, Fernández GB, Peck GS. Management of hepatic trauma: four years experience. [Article in Spanish] *Cir Esp* 2011;89:511-6. [Abstract]
21. Tan KK, Bang SL, Vijayan A, Chiu MT. Hepatic enzymes have a role in the diagnosis of hepatic injury after blunt abdominal trauma. *Injury* 2009;40:978-83. [CrossRef](#)
22. Clarke JR, Trooskin SZ, Doshi PJ, Greenwald L, Mode CJ. Time to laparotomy for intra-abdominal bleeding from trauma does affect survival for delays up to 90 minutes. *J Trauma* 2002;52:420-5. [CrossRef](#)

23. Wilson RH, Moorehead RJ. Hepatic trauma and its management. *Injury* 1991;22:439-45. [CrossRef](#)
24. Sauaia A, Moore FA, Moore EE, Moser KS, Brennan R, Read RA, et al. Epidemiology of trauma deaths: a reassessment. *J Trauma* 1995;38:185-93. [CrossRef](#)
25. Gruen RL, Jurkovich GJ, McIntyre LK, Foy HM, Maier RV. Patterns of errors contributing to trauma mortality: lessons learned from 2,594 deaths. *Ann Surg* 2006;244:371-80.
26. Brohi K, Cohen MJ, Ganter MT, Matthay MA, Mackersie RC, Pittet JF. Acute traumatic coagulopathy: initiated by hypoperfusion: modulated through the protein C pathway? *Ann Surg* 2007;245:812-8. [CrossRef](#)
27. Cosgriff N, Moore EE, Sauaia A, Kenny-Moynihan M, Burch JM, Galloway B. Predicting life-threatening coagulopathy in the massively transfused trauma patient: hypothermia and acidosis revisited. *J Trauma* 1997;42:857-62. [CrossRef](#)
28. Brohi K, Singh J, Heron M, Coats T. Acute traumatic coagulopathy. *J Trauma* 2003;54:1127-30. [CrossRef](#)
29. MacLeod JB, Lynn M, McKenney MG, Cohn SM, Murtha M. Early coagulopathy predicts mortality in trauma. *J Trauma* 2003;55:39-44.
30. Martini WZ, Dubick MA, Pusateri AE, Park MS, Ryan KL, Holcomb JB. Does bicarbonate correct coagulation function impaired by acidosis in swine? *J Trauma* 2006;61:99-106. [CrossRef](#)
31. Frith D, Davenport R, Brohi K. Acute traumatic coagulopathy. *Curr Opin Anaesthesiol* 2012;25:229-34. [CrossRef](#)
32. Brown LM, Call MS, Margaret Knudson M, Cohen MJ. (Trauma Outcomes Group). A normal platelet count may not be enough: the impact of admission platelet count on mortality and transfusion in severely injured trauma patients. *J Trauma* 2011;71(2 Suppl 3):337-42. [CrossRef](#)
33. Croce MA, Fabian TC, Menke PG, Waddle-Smith L, Minard G, Kudsk KA, et al. Nonoperative management of blunt hepatic trauma is the treatment of choice for hemodynamically stable patients. Results of a prospective trial. *Ann Surg* 1995;221:744-55. [CrossRef](#)
34. Domínguez Fernández E, Aufmkolk M, Schmidt U, Nimitz K, Stöblen F, Obertacke U, et al. Outcome and management of blunt liver injuries in multiple trauma patients. *Langenbecks Arch Surg* 1999;384:453-60.
35. Ritchie AH, Williscroft DM. Elevated liver enzymes as a predictor of liver injury in stable blunt abdominal trauma patients: case report and systematic review of the literature. *Can J Rural Med* 2006;11:283-7.

KLİNİK ÇALIŞMA - ÖZET

Üçüncü basamak hastanede karaciğer travmalarının değerlendirilmesi: Geriye dönük bir çalışma

Dr. İsmail Bilgiç,¹ Dr. Sibel Gelecek,¹ Dr. Ali Emre Akgün,¹ Dr. Mehmet Mahir Özmen²

¹Ankara Numune Eğitim ve Araştırma Hastanesi, Genel Cerrahi Kliniği, Ankara;

²Hacettepe Üniversitesi Tıp Fakültesi, Genel Cerrahi Kliniği, Ankara

AMAÇ: Abdominal travmalarda karaciğer en sık yaralanan organdır. Politravmatik bir hastada karaciğer yaralanması minör yaralanmalardan majör yaralanmalara kadar değişen bir şekilde etkilenebilir. Bu yaralanma derecesine göre morbidite ve mortaliteye katkıda bulunur. Bu çalışmanın amacı ameliyatta karaciğer yaralanması tespit edilen hastaların sonuçlarının değerlendirilmesidir.

GEREÇ VE YÖNTEM: 2005 ve 2013 yılları arasında, sadece cerrahi sırasında karaciğer hasarı tespit edilen 82 hasta geriye dönük olarak incelendi. Hastaların demografik verileri, laboratuvar verileri, ameliyat bulguları ve yapılan ameliyatları incelendi. Hastalar iki gruba ayrıldı: Mortalite grubu ve yaşayan hastalar grubu.

BULGULAR: Çalışmadaki erkek hasta sayısı 77 (%94), kadın hasta sayısı beş (%6) olarak bulundu. Ortalama yaş 34 (17-90) idi; 15 (%18.3) hastada mortalite gözlemlendi, 34 (%41.5) hastada künt karın travması, 48 (%48.5) hastada penetran yaralanma mevcuttu. Hastaların 47'sinde (%57) çoklu travma mevcuttu, 47 (%57) hastada 71 adet eşlik eden karınıci diğer organ yaralanmaları mevcuttu. Başvuru anında hemodinamik olarak 46 (%56.1) hasta stabil, 36 (%43.9) hasta instabil idi. Başvuru anındaki AST, ALT, LDH, APTT, PT, INR ve kreatinin değerleri mortalite grubunda yaşayan hasta grubuna göre daha yüksek, fibrinojen ve trombosit sayısı ise daha düşük idi.

TARTIŞMA: Hemodinamik instabilite, eşlik eden iskelet sistemi göğüs travması, başvuru anındaki yüksek APTT, PT, INR, AST, ALT, LDH ve düşük fibrinojen ve trombosit değerleri mortalite açısından prognostik faktörler olabilir.

Anahtar sözcükler: Karaciğer enzimleri; karaciğer hasarı; mortalite.

Ulus Travma Acil Cerrahi Derg 2014;20(5):359-365 doi: 10.5505/tjtes.2014.22074