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Transarterial Embolization Affects Recovery in Nonoperative Management of Severe Blunt Hepatic Injuries: A Retrospective Study

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Abstract

Introduction: Transarterial embolization (TAE) has been used as an essential technique in the nonoperative management (NOM) of severe blunt hepatic injuries (BHI) but has a possible risk of tissue ischemia and necrosis, which may affect hepatic recovery. The injured liver tissue gets encapsulated and absorbed by intact liver tissue; therefore, the healing process requires the formation of a wall surrounding the injured tissue. In this study, we determined the impact of TAE on the hepatic healing process.

Patients and methods: We reviewed hemodynamically stable patients (n=35; males, 23; females, 12; mean age, 32 years), admitted to our hospitals over a 10-year period, who underwent NOM of severe BHI (American Association for the Surgery of Trauma Organ Injury Scale, grades IV and V). Data collected included demographic information, serial CT findings, severity of hepatic injuries showing lacerations (Couinaud's liver segment classification), TAE performed for active hepatic bleeding, and the amount of hemoperitoneum. Extrahepatic factors were evaluated using the Abbreviated Injury Scale for injuries to the head, face, thorax, and extremities. Time taken by injured segments to encapsulate was individually obtained from serial CTs. Cox regression and Kaplan-Meier analyses were used to identify risk factors associated with delayed healing among the hepatic and extrahepatic factors mentioned above.

Results: Risk factors were independently and significantly associated with TAE (odds ratio, 2.45; 95% confidence interval, 1.01-5.92; p=0.047). Multivariate analysis indicated that patients who underwent TAE took 2.45-times longer time to attain liver encapsulation than those who did not undergo TAE and that extrahepatic factors did not correlate with the time to encapsulation.

Conclusion: TAE for active liver hemorrhage resulted in a delayed recovery from BHI, regardless of extrahepatic factors.

Keywords: TAE; Liver injury; Encapsulation; Healing process; Blunt trauma; Biloma

Introduction

Transarterial embolization (TAE) has been used as an essential technique for the treatment of blunt hepatic injuries (BHI) since nonoperative management (NOM) became the standard of care for hemodynamically stable patients with BHI [1-3]. Several studies have confirmed the feasibility and safety of NOM for severe BHI, and TAE remains the treatment of choice for severe BHI [4]. While the use of TAE in the initial management of severe BHI has increased, several complications after TAE have been reported [5-7]. Among these, delayed hemorrhage from hepatic injuries is rare but potentially life-threatening for patients with BHI in the acute phase of injury [8-10], whereas biliary peritonitis and perihepatic abscess may be critical at a later phase.

In our general experience, severe BHI spontaneously heal, irrespective of TAE; however, the results of NOM may partly depend on the healing process, including encapsulation and absorption of the injured liver tissue surrounded by intact liver or other tissue, which requires the formation of a wall surrounding the injured liver tissue. However, TAE has a possible risk of tissue necrosis, which may affect the healing process; therefore, the impact of TAE on the healing process should not be ignored. In this study, we determined the impact of TAE on the healing process in patients with severe BHI who underwent TAE.

Patients and Methods

Patient management and CT scanning

Over a 10-year period (January 2000-December 2010), we reviewed all patients with severe BHI admitted to our trauma centers, which include the Keio University Hospital (Tokyo, Japan), Saiseikai Kanagawaken Hospital (Yokohama, Japan), and Saiseikai Yokohamashi Tobu Hospital (Yokohama, Japan). The trauma center at the Saiseikai Kangawaken Hospital was moved to the Saiseikai Yokohamashi Tobu Hospital in March 2006. Severe BHI was defined as AAST-OIS [11] grades IV and V (Table 1). All clinical data including vital signs, blood tests, and radiographic and CT findings were obtained by reviewing patient charts and imaging studies. The severity of BHI was assessed by the area lacerated (Couinaud's liver segment classification), performance of TAE for active hepatic bleeding, and amount of

hemoperitoneum. Extrahepatic factors were evaluated using the Abbreviated Injury Scale (AIS) for injuries to the head, face, thorax, and extremities and the Injury Severity Score (ISS). Time taken by injured segments to encapsulate was individually obtained from serial CT scans. Cox regression and Kaplan–Meier analyses were used to identify risk factors associated with delayed healing.

Grade	Type of Injury	Description
I	Hematoma	Subcapsular, nonexpanding, <10% surface area
	Laceration	Capsular tear, nonbleeding, <1 cm parenchymal depth
II	Hematoma	Subcapsular, nonexpanding, 10% to 50% surface area
	Laceration	Capsular tear, nonbleeding, <1 cm parenchymal depth
III	Hematoma	Subcapsular, >50% surface area or expanding; ruptured subcapsular hematoma with active bleeding; intraparenchymal hematoma >10 cm or expanding
	Laceration	>3 cm in depth
IV	Hematoma	Ruptured intraparenchymal hematoma with active bleeding
	Laceration	Parechymal disruption involving 25% to 75% of a hepatic lobe or one to three Couinaud segments within a single lobe
V	Laceration	Parenchymal disruption involving >75% of a hepatic lobe or more than three Couinaud segments within a single lobe
	Vascular	Juxtahepatic venous injuries (i.e., retrohepatic vena cava or central hepatic veins)
VI	Vascular	Hepatic avulsion

Table 1: Liver injury scale.

Hemodynamically stable patients with severe BHI after fluid resuscitation in our emergency department were selected for NOM. Our criteria for NOM of BHI included hemodynamic stability or an immediate response to crystalloid resuscitation and the absence of other intraabdominal injuries requiring surgery. Patients presenting with hemodynamical deterioration during the early phase after admission underwent laparotomy and were excluded from this study. Hemodynamically stable patients with severe BHI were closely monitored in the ICU to check for signs or symptoms of any cardiopulmonary complication for at least 2 d. If the hemodynamics remained stable, then patients were placed in a general ward.

BHI was repeatedly evaluated by several imaging examinations by CT scans and focused assessment with sonography for trauma patients (FAST) until their BHI were encapsulated by a ≥ 5-mm parenchymal wall as demonstrated in the follow-up CT scans. All CT scans were interpreted by staff radiologists in our own hospitals, and no oral contrast medium was used. When the initial CT findings indicated active bleeding, a large and lacerated area of three or more segments, pseudoaneurysma, or arterioportal shunt formation or when hemoperitoneum increased as measured by FAST, patients underwent angiography.

TAE was performed if angiography revealed continuous arterial bleeding or a pseudoaneurysm associated with hepatic injury. During the study period, superselective TAE for severe BHI was performed by the same radiologist, and the criteria for TAE did not change, except for small adjustments (material and instrument improvements). Prophylactic antibiotics before and after TAE were routinely administered for 1 d. The amount of associated hemoperitoneum was estimated based on the criteria established by Federle and Jeffrey [12].

To observe the healing process of BHI with or without TAE, initial and follow-up CT scans were routinely performed at a predetermined time interval. The initial evaluation of BHI by CT was performed on ER arrival (day 1), and at least one or more imaging examinations by FAST or CT were repeated in the first 3 to 4 d after injury. Thereafter, follow-up CT scans were performed approximately every week (7 ± 2 d) until the injured liver tissue was completely encapsulated by a ≥ 5-mm parenchymal wall. Based on our general experience, the healing process requires the formation of a wall surrounding the injured liver tissue. In this study, a 5-mm wall was considered as the cornerstone of the healing process, and it was assessed by reviewing CT scans. The interval between imaging studies depended on each patient's condition. Additional CT scans were immediately taken if a patient with BHI showed any sign or symptom suggestive of serious complications. If necessary, a FAST examination was performed between weekly CT scan to avoid excessive X-ray exposure.

Statistical analysis

The time taken by injured liver segments to encapsulate was individually obtained from serial CT findings. Comparative analysis of categorical variables was performed using the X2 test with Yate's correction for continuity. Continuous variables were analyzed using Student's t-test for normally distributed variables; otherwise, the Mann–Whitney U test was employed. Independent variables with a p-value of <0.2 by univariate statistics were included in the multivariate analysis.

A multivariate Cox proportional hazards regression model was developed to test the association between TAE and time to encapsulation. Kaplan–Meier analysis was used to demonstrate risk factors associated with delayed healing. Values are expressed as mean ± SD. The results of the Cox regression are reported as hazard ratios with 95% confidence intervals. All p-values are two-tailed, and $p<0.05$ was considered statistically significant. All statistical analyses were performed using SPSS software (version 14.0J, SPSS Inc.).

Ethics

This study was approved by the research and ethics committees at the authors' respective hospitals.

Results

Patient demographics and injury characteristics

During the study period, 72 patients with severe BHI presented at the above-mentioned hospitals, of which 34 with hemodynamical instability underwent urgent laparotomy and the remaining 38 were all initially selected for NOM; however, three patients with hemodynamic deterioration in the acute phase underwent emergency surgery and were excluded (their surgeries identified hepatic vein injuries as the cause of bleeding). Finally, 35 patients (males, 23; females, 12; mean

age, 32 years) were hemodynamically stable and admitted to the ICU for NOM.

Their injuries resulted from motorcycle crashes (12 patients), motor vehicle crashes (seven patients), pedestrian accidents (seven patients), bicycle accidents (four patients), falls (two patients), assault (one patient), and other causes (two patients). Of the 35 patients, 14 underwent TAE. Univariate analysis indicated that patients with BHI who underwent TAE had significantly more lacerated liver segments and contrast extravasation on CT (Table 2) and that there was no significant difference in either the rate of AAST-OIS grade V or amount of hemoperitoneum between patients who underwent TAE and those who did not. There was no significant difference in the AIS scores for anatomic extrahepatic factors, including injuries to the head, face, thorax, and extremities, and in ISS between the two groups. Patients who underwent TAE had a greater number of transfusions, longer ICU/hospital stays, and a longer time to encapsulation than those who did not. Overall, NOM failed in two patients, and one patient died. No enteric injuries were missed during NOM of patients with severe BHI.

TAE delayed hepatic healing of severe BHI

In most cases of patients with severe BHI, injured liver tissue spontaneously healed, irrespective of TAE. Representative cases are shown in Figures 1 and 2. As shown in Figure 1, contrast-enhanced helical CT of the abdomen showed grade IV BHI with contrast blush and pooling in the liver (Figures 1A and 1B). Hepatic angiography indicated a pseudoaneurysm with extravasation in the liver (Figures 1C and 1D). After angioembolization was successfully performed by microcoil at a selected branch of A5, follow-up CT scans demonstrated that injured liver tissue was completely encapsulated within a 5-mm wall on day 25 (Figures 2A and 2B), and the damaged area gradually shrunk afterward (Figure 2C). Based on this observation, we assessed the healing process in patients with severe BHI with and without TAE.

Variables		**TAE +** **(n=14)**	**TAE −** **(n=21)**	**p**
Age (mean)		37.4 ± 22.5	27.6 ± 16.6	0.15
Male, gender, n (%)		10 (71.4)	13 (61.9)	0.81
SBP on ER arrival (mmHg)		109 ± 26	116 ± 20	0.41
AAST-OIS, grade V, n (%)		3 (21.4)	1 (4.8)	0.32
Number of liver segments lacerated		2.8 ± 1.0	2.1 ± 0.8	0.03
Extravasation observed on CT, n (%)		6 (42.8)	0	0.004
Moderate or large HP on ER arrival, n (%)		5 (35.7)	4 (19.0)	>0.99
Conversion to operative management, n (%)		2 (14.2)	0	0.12
AIS	head	0.9 ± 1.6	0.3 ± 0.9	0.17
	face	0.4 ± 0.5	0.5 ± 0.8	0.62
	thorax	2.0 ± 1.7	1.0 ± 1.4	0.052
	abdomen	4.4 ± 0.5	4.7 ± 0.5	0.09
	extremities	1.0 ± 1.2	1.0 ± 1.2	0.91
ISS		32 ± 11	28 ± 8	0.27
Transfusion (U)		10.5 ± 11.2	1.5 ± 3.8	0.0016
ICU stay (days)		20 ± 29	4 ± 4	0.03
Time course to encapsulation (days)		23 ± 15	14 ± 6	0.01
Mortality, n (%)		1 (7.1)	0	0.81

Abbreviations: TAE: Transarterial Embolization; HR: Heart Rates; ER: Emergency Room; CT: Computed Tomography; HP: Hemoperitoneum; AAST-OIS: The American Association for the Surgery of Trauma Organ Injury Scale; AIS: The Abbreviated Injury Scale; ISS: Injury Severity Score; ICU: Intensive Care Unit.

Table 2: Patient demographics and injury characteristics.

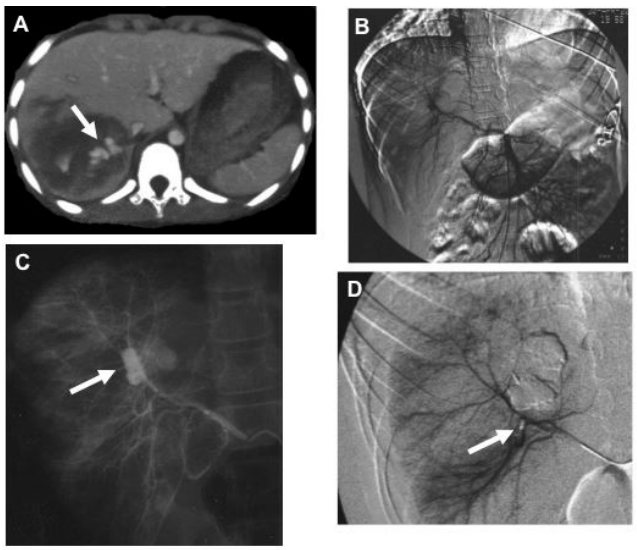

Figure 1: Transarterial embolization for pseudoaneurysm (branch in A5) and extravasation in severe blunt hepatic injuries. An 18-year-old female with severe blunt hepatic injury. (A) Contrast-enhanced helical CT scan of the abdomen showed contrast blush and pooling in the liver (arrows). Hepatic angiography indicated that right hepatic artery replacement from supramesenteric artery (B) and a pseudoaneurysm with extravasation in the liver (arrow) (C). (D) Angioembolization was performed successfully by microcoil at a selected branch of A5.

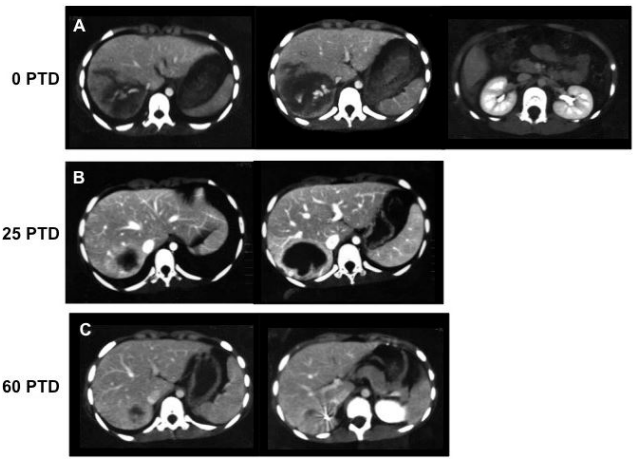

Figure 2: Healing process of severe blunt hepatic injuries to encapsulation. Follow-up CT scans of patient depicted in Figure 1. (A) Initial CT scans of patient before angioembolization. (B) CT scans performed 25 days after angioembolization demonstrating encapsulated form of biloma in the injured liver tissue. (C) shrinking of encapsulated biloma 60 days after angioembolization.

Analysis of time course to encapsulation

Among the 35 patients, multivariate analysis indicated that patients who underwent TAE took a significantly longer time to attain liver encapsulation (Table 3). Extrahepatic factors such as injuries to the head, face, thorax, and extremities (AIS scores) and the degree of hemoperitoneum did not correlate with the time to encapsulation.

Variable	Hazard Ratio	95% Confidence Interval	p
TAE	2.45	1.01-5.92	0.047
Segments	0.59	0.33-1.08	0.088
Abbreviations: TAE: Transarterial Embolization			

Table 3: Results for multivariate cox regression analysis time course to encapsulation.

It took 2.45-times longer time to attain encapsulation for patients who underwent TAE than for those who did not (Figure 3). Interestingly, in the healing process, the slopes of lines for encapsulation rates appeared to be similar in patients who underwent TAE and in those who did not. In patients with severe BHI who did not undergo TAE, the rates of encapsulation formation were 19.0% (1 week), 57.1% (2 w), and 90.5% (3 w), whereas in those who underwent TAE, these were 0.0% (1 w), 28.6% (2 w), and 57.1% (3 w). There was approximately a 1-week delay in the time to encapsulation between the two groups.

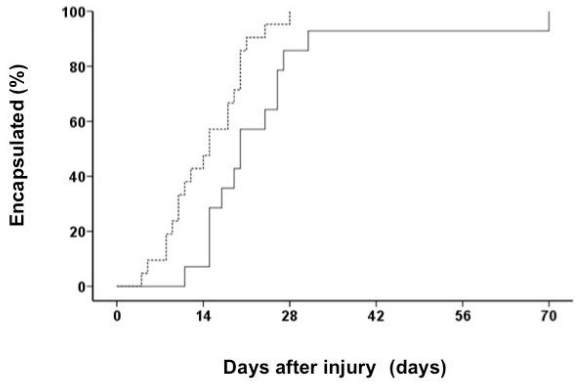

Figure 3: Time course of severe blunt hepatic injuries to encapsulation. All hemodynamically stable patients with severe blunt hepatic injuries (BHI) were evaluated by serial follow-up CT scans on a routine basis at a predetermined time interval until the BHI were fully encapsulated by a ≥ 5-mm parenchymal wall. In Cox regression models, TAE was significantly related to longer duration for encapsulation. The time courses of healing to encapsulation are shown for BHI patients who underwent TAE and those who did not. A solid line represented the BHI patients who underwent TAE, whereas a dashed line represented those who did not. The beginning of encapsulation was delayed in the former. At the beginning of the healing process, the slopes of the encapsulation rates appeared to be similar in patients with and without TAE. Between the two patient groups, there was about one-week delay to encapsulation of the injuries.

Complications

Patients who did not undergo TAE had non-liver-related complications (including fever in three and jaundice, with an increase of indirect bilirubin, in one), but they did not experience any liver-related complication. In contrast, several patients who underwent TAE had serious complications: early-phase respiratory failure in three (prior to day 3); perihepatic infections in two, including a liver abscess in one (day 14); and acute, late-phase, gangrenous cholecystitis in one (day 64). Two patients with serious infectious complications underwent surgery for infection control and drainage, despite numerous attempts at percutaneous drainage. The patient with liver abscess underwent hepatectomy following tissue necrosis on day 14 and uneventfully recovered, whereas the patient with acute, late-phase, gangrenous cholecystitis who underwent cholecystectomy on day 64 died of multiple organ dysfunction syndrome on day 116.

Discussion

We aimed to review the cases of patients with severe BHI to assess the impact of TAE in arresting liver hemorrhage on the hepatic healing process. In this study, multivariate Cox regression analysis indicated that TAE was identified as an independent risk factor for the delayed encapsulation of injured liver tissue among several hepatic and extrahepatic factors associated with liver injuries in hemodynamically stable patients with severe BHI. Considering that major hepatic necrosis is a common complication after TAE in hemodynamically unstable patients with severe BHI [7], the hepatic healing process depends on both hepatic local blood flow and systemic circulation. However, TAE for severe BHI stopped continuous arterial hemorrhage in an injured liver, which otherwise would have required an emergency surgery with its associated high morbidity and mortality. Thus, we believe that the delayed healing caused by TAE is a clinically acceptable complication in patients with severe BHI after TAE. There are two possible explanations for delayed healing in patients who underwent TAE.

Focal liver damage is restored by a mechanism resembling that for wound healing [13]. This mechanism is responsible for the proliferation of hepatocytes surrounding the damaged sites [13], and blood supply and oxygen delivery are important for tissue regeneration. In patients with severe BHI with injury to intrahepatic portal branches, TAE performed to arrest hepatic arterial hemorrhage may induce a serious decrease in the blood supply of and oxygen delivery to the liver.

Among the other factors affecting the healing process of hepatic injuries, bile is reported to be associated with the impaired healing of liver tissue as well as delayed bleeding and pseudoaneurysm formation [14,15]. Bile discharge diminishes fibrinous exudate production and granulation tissue and fibrous scar formation [14]. Previous reports have demonstrated a much higher incidence of biloma in patients with severe BHI after TAE (29.6%, 8/27) than in those with severe BHI overall (5.8%; 11/189) [15,16]. In patients with severe BHI, a subsequent biloma formation may cause a delayed healing of liver injuries after TAE. While TAE is essential for arresting hepatic arterial hemorrhage, it can also induce liver ischemia (necrosis) along with biloma formation, leading to delayed healing. Superselective embolization is recommended to prevent this adverse effect.

Interestingly, TAE appeared to be associated with a delayed initiation of liver healing, but in the healing process, the slopes of lines for encapsulation rates appeared to be similar in patients who underwent TAE and in those who did not, suggesting that the time to encapsulation mainly depends on arterial blood supply and that there is a limited impact of TAE on the acute injury phase (<1 week after injury) in cases of patients with severe BHI.

Complications related to TAE

All 35 patients who underwent TAE had no delayed hemorrhage or pseudoaneurysm ruptures related to their BHI. Of the 14 patients with BHI after TAE, two underwent surgery for liver-related complications. These complications were more frequent in patients with BHI who underwent TAE than in those who did not. Liver-related complications in NOM of severe BHI have been described in several reports. Except for the initial hemorrhagic events during the early phase of NOM, delayed complications were primarily biliary and infectious [1,5,6,16]. As the two cases of infectious complications occurred ≥ 2 weeks after injury, the susceptibility of infection in patients with severe BHI after TAE may comprise retrograde bacterial contamination from the biliary tree and immunoparalytic host response following severe injuries. Compared with previous reports, we found an extremely low incidence of delayed hemorrhage in our patients with severe BHI (0%-14%) [6,16,17]. Superselective embolization for patients with severe BHI may have contributed to this low incidence.

Follow-up CT scans of severe BHI

During the initial phase of liver trauma, CT scans significantly contributed to anatomic evaluations and clinical decisions regarding NOM of severe BHI in hemodynamically stable patients. Previous reports have concluded that follow-up CT scans did not change decision-making for patients with grades I–III BHI [18]. Furthermore, CT scans should be indicated only for patients with any grade of BHI who develop signs or symptoms suggestive of hepatic complications [19,20]. Our results showed that all patients with grade IV/V BHI who did not undergo TAE had no hepatic complications and displayed excellent healing within 3 weeks (90.5%), whereas those who underwent TAE had a delay in the hepatic recovery by 1 week. Two patients who underwent TAE suffered from hepatic infectious complications with delayed healing, but our protocol of weekly CT scans did not predict serious infectious complications in patients with severe BHI after TAE. Thus, we do not recommend routine follow-up scans in patients with severe BHI who have no signs or symptoms suggestive of infectious complications and in patients with low-grade BHI. We suggest that cautious management is necessary for patients with severe BHI who underwent TAE because TAE in severe BHI was identified as an independent risk factor for delayed healing.

Conclusion

TAE for active liver hemorrhage resulted in a delayed recovery in patients with BHI, regardless of extrahepatic factors.

Competing interests

The authors declare that they have no conflict of interests.

Author's Contributions

KS, SM, TF, and YS contributed to the collection, analysis, and interpretation of data and drafting of the manuscript. JS and MK provided the critical review of this manuscript. All authors have read and approved the final manuscript.

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