Nonsurgical Management of Patients with Blunt Hepatic Injury: Efficacy of Transcatheter Arterial Embolization

Akiyoshi Hagiwara[1]
Tetsuo Yukioka
Shoich Ohta
Takahiko Tokunaga
Shin Ohta
Hiroharu Matsuda
Shuji Shimazaki

OBJECTIVE. We evaluated the efficacy of transcatheter arterial embolization (TAE) for patients with blunt hepatic injury.

SUBJECTS AND METHODS. Of 372 patients with trauma, 60 had evidence on CT of hepatic injury (Mirvis classification). Six of the 60 patients required emergency laparotomy and were excluded. Of the 54 remaining patients, 28 were classified as having high-grade hepatic injury (Mirvis classification of 3 or 4). All 28 underwent arteriography, and TAE was performed in single or multiple hepatic arterial branches when extravasation was seen. Angiography was repeated and cholescintigraphy was performed on patients with continued bleeding or biloma.

RESULTS. Injuries detected were grade 1 (n = 13), grade 2 (n = 13), grade 3 (n = 20), and grade 4 (n = 8). The injury was correlated with the degree of hemoperitoneum seen on CT. Patients with low-grade injuries (Mirvis classification of 1 or 2) were treated conservatively, and no deaths or liver-related morbidity occurred. Of the 28 patients with high-grade injury, 15 also had angiographic evidence of extravasation and underwent TAE. The average fluid resuscitation volume was significantly larger in this group than in the other 13 patients with high-grade injuries who did not undergo TAE. Embolization was successful in all 15 patients, and the shock index was significantly reduced after TAE. All patients survived, with follow-up at 1–8 months (2.5 ± 1.8 months, mean ± SD).

CONCLUSION. TAE is an effective alternative to surgery for patients with high-grade liver injury.

Nonsurgical management has been shown to be effective for patients with mild hepatic injury (hepatic injury of abbreviated injury scale 1 or 2 [1], with hemodynamic stability) [2–7]. However, this therapy has not been as effective for patients with severe hepatic injury in whom surgery is often required. A major problem for management of the latter patients is that CT imaging fails to provide information about vascular injuries of the liver that may be responsible for hemorrhage and is critical for achieving early hemostasis. Recently, transcatheter arterial embolization (TAE) has been shown to be effective for patients with blunt hepatic trauma and subacute hepatic rupture [8, 9], and one study has shown that emergency TAE may be effective for unstable patients with severe blunt hepatic injury [10]. We studied patients with blunt hepatic injury and investigated the use of angiography and TAE in patients with high-grade injuries.

Subjects and Methods

A prospective clinical study was performed at our institution from January 1992 to May 1996. After initial examination and resuscitation, all patients with blunt abdominal injury who were hemodynamically stable, with or without fluid resuscitation, underwent CT imaging of the abdomen and pelvis. A solution of 62% iopamidol (Iopamiron 300; Nihon Schering, Osaka, Japan) was administered as an IV bolus injection of 15–50 ml at a rate of 1 ml/sec, unless contraindicated by a history of allergy to contrast material or serum creatinine concentration of greater than 2.5 mg/dl. The scanning was performed in 1-cm-thick sections from the diaphragmatic dome to the pelvis using a CT 9200 scanner (General Electric Yokogawa Medical Systems, Tokyo, Japan). CT findings of hepatic injury were evaluated according to the classification of Mirvis et al. [6] (Table 1). All patients with CT evidence of hepatic injury were considered for the protocol; however, the following were excluded: patients who required emergency surgery for reasons other than hepatic injury (e.g., intestinal perforation or pancreatic injury), patients who were hemodynamically unstable and did not

Received December 16, 1996; accepted after revision April 12, 1997.

[1] All authors: Department of Traumatology and Critical Care Medicine, Kyorin University School of Medicine, 6-20-2 Shinkawa Mitaka-Si Tokyo 181, Japan. Address correspondence to A. Hagiwara.

AJR 1997;169:1151–1156

0361–803X/97/1694–1151

TABLE 1 CT-Based Injury Severity Grades for Blunt Hepatic Trauma

Grade	Criteria
1	Capsular avulsion, superficial laceration(s) <1 cm deep, subcapsular hematoma <1 cm maximal thickness, periportal blood tracking only
2	Laceration(s) 1–3 cm deep, central or subcapsular hematoma(s) 1–3 cm in diameter
3	Laceration(s) >3 cm deep, central or subcapsular hematoma(s) >3 cm in diameter
4	Massive central or subcapsular hematoma >10 cm, lobar tissue destruction (maceration) or devascularization
5	Bilobar tissue destruction (maceration) or devascularization

Note.—Data taken from [6].

improve with fluid resuscitation, and patients who had other severe visceral injury (e.g., splenic injury, renal injury, or mesenteric injury) and in whom massive bleeding was suspected. Patients with hepatic injury classified on CT as grade 3 or greater were referred for angiography.

Patients included in the protocol underwent angiography (cut-film or digital subtraction) within 3 hr of the initial CT scan. An abdominal aortogram or celiac arteriogram was obtained after administration of 76% iopamidol (Iopamiron 370; Nihon Schering) at a rate of 5–20 ml/sec (for a total of 20–40 ml). Next, a hepatic arteriogram was obtained after administration of 76% iopamidol at a rate of 2–5 ml/sec (for a total of 10–20 ml). The time allotted for angiography was limited to 1 hr. The maximum contrast material load was limited to 150 ml.

Patients were divided into four groups on the basis of angiographic findings: group A, variable avascularity and irregularity in the accumulation of contrast medium in the hepatic parenchyma or displacement of the hepatic artery branches; group B, hepatic artery–portal vein fistula or disruption of hepatic artery branches; group C, extravasation of contrast medium extending within the hepatic parenchyma; and group D, extravasation of contrast medium extending beyond the hepatic parenchyma. Transcatheter embolization was performed for all patients with group C or D findings.

Our technique for TAE was as follows. For extravasation of a single hepatic artery, we tried to insert a standard 5-French catheter into the injured artery using a standard guidewire technique. If this technique failed, we used a 3-French microcatheter (Tracker-18 Unibody infusion catheter; Target Therapeutics, Fremont, CA) through a standard 5-French guiding catheter. Gelatin sponge particles were used as the embolic material. For extravasation from more than one hepatic artery, gelatin sponge particles were first dispersed through a catheter positioned distal to the cystic artery; when extravasation persisted after use of more than one gelatin sheet, stainless-steel coils (embolization coil MWCE-35-4-3, MWCE-35-5-5, or MWCE-35-5-8 [Cook, Bloomington, IN] or MWCE embolization microcoil MWCE-18S-2.4-4-A or MWCE-18S-5-B [Cook, Bjaeverskov, Denmark]) were deployed in a proximal position in the extravasating arteries (e.g., segmental artery, right, middle, and left artery). Stasis of contrast material in the proximal hepatic artery was used as the end point for the termination of coil delivery. Finally, celiac or hepatic arteriography was repeated to confirm that no residual extravasation of the hepatic artery was evident.

All patients with hepatic injury were admitted to the surgical intensive care unit for clinical observation. Physical examination and monitoring of vital signs were repeated at short intervals. Angiography was performed immediately when patients had findings of ongoing hemorrhage or rehemorrhage from the injured liver (i.e., increasing intraabdominal hemorrhage or intrahepatic hematoma revealed on sonography or CT). A provision for suspension of the protocol was made for any patient who showed signs of peritonitis and required an emergency laparotomy.

Contrast-enhanced CT was repeated on days 7, 14, and 21 after admission and at monthly intervals until the hematoma disappeared. ^{99m}Tc-Pyridoxyl-5-methyl-tryptophan cholescintigraphy was performed in patients who were suspected of having biloma (i.e., patients with a persistent or increasing localized low-density area in the injured liver [11] on CT at day 7). Angiography was repeated in patients who were suspected of rebleeding from the injured liver during hospitalization (e.g., increasing intrahepatic hematoma or intraabdominal hemorrhage visualized on sonography or CT) and patients in whom the cholescintigram showed a biloma.

This study was approved by the Human Subjects Committee at our institution. Informed consent was obtained from each patient (or guardian).

Data are expressed as mean ± SD. Differences between groups were analyzed using the Student's *t* test. The CT injury grade was correlated with the degree of hemoperitoneum seen on CT, using the Kruskal-Wallis test. A *p* value of less than .05 was considered significant.

Results

Of the 372 patients admitted with blunt abdominal trauma, 60 (16%) showed CT evidence of hepatic injury. They included 18 women and 42 men who were 3–75 years old (28.7 ± 16.9 years). The injury severity score [12] ranged from 4 to 41 (16.9 ± 9.2). The injuries were caused by motor vehicle accidents (n = 32), pedestrian–automobile accidents (n = 9), falls (n = 7), and assault (n = 12).

Six of the 60 patients underwent emergency laparotomy and were excluded from this study. Four of these six patients had severe associated injuries. Two other patients became suddenly hypotensive after CT and required emergency laparotomy. One of these two had a CT grade 3 injury accompanied by disruption of the right major hepatic vein; this patient underwent a right hepatic lobectomy but died because of ongoing coagulopathy after laparotomy. The other patient had a CT grade 4 injury and underwent a right hepatic segmentectomy; he recovered uneventfully after laparotomy and was discharged in good condition after 69 days. The remaining 54 patients were enrolled in the protocol.

The CT grade and degree of hemoperitoneum for 54 patients enrolled in the protocol are shown in Table 2. CT grade 3 injuries were the most frequent. No patients had CT grade 5 injuries. The CT grade was correlated with the degree of hemoperitoneum.

Angiography was performed in 28 patients with hepatic injury equal to or greater than CT grade 3. The angiographic findings for patients with various CT grades are summarized in Table 3. In all eight patients with CT grade 4 injuries, extravasation of contrast medium was seen. TAE was performed in all 15 patients with group C or D angiographic findings (Figs. 1 and 2). TAE was successful in all 15 patients, and no extravasation was seen on celiac or hepatic arteriograms obtained after embolization. All 15 patients were hemodynamically stable after TAE. One patient underwent explor-

TABLE 2 CT Hepatic Injury Severity Grades and Degree of Hemoperitoneum in 54 Patients with Blunt Abdominal Trauma

Hemo.	CT Grade[a]				
	1 (13)	2 (13)	3 (20)	4 (8)	5 (0)
0	13	10	5	0	0
1+	0	3	5	0	0
2+	0	0	6	5	0
3+	0	0	4	3	0

Note.—Hemo. = hemoperitoneum. Hemoperitoneum is quantified as 0 = none detected; 1+ = confined to a single anatomic area such as Morison's pouch (hepatorenal space), perihepatic space, or perisplenic space; 2+ = in two or more anatomic spaces such as the pericolic gutter and lateral perivesicle space; 3+ = in entire pelvis.

[a]Number of patients is given in parentheses.

atory laparotomy after TAE to evaluate a soft-tissue density in the left upper abdominal quadrant seen on CT images (Fig. 2B), which was found to be a large hematoma in the omentum. At surgery, the liver initially showed no bleeding. Almost immediately, however, an uncontrollable massive hemorrhage from the liver occurred, and the patient became severely hypotensive. Large amounts of gauze were packed around the injured liver, and angiography was repeated immediately after closure (Figs. 2E and 2F). Extravasation of contrast medium was seen from multiple hepatic artery branches, and TAE was performed again. The patient became hemodynamically stable after embolization, and the gauze was removed 3 days later. Within 3–6 months, all patients with hepatic injury of CT grade 3 or greater showed resolution of the low-density areas in the liver on CT scans.

Patients with CT grade 1 or 2 injuries did not undergo angiography and were treated with conservative management; they recovered uneventfully without liver-related complications. CT scans of all such patients on day 7 (after admission) showed decreasing localized low-density areas within the hepatic parenchyma. These low-density areas were no longer evident on CT images obtained 1 month after injury.

The shock index and average volume of fluid resuscitation for patients who underwent TAE and those who did not are shown in Table 4. The shock index was significantly lower after TAE ($p < .001$). The average volume of fluid resuscitation required during the 3 hr after admission was significantly lower for patients who did not undergo TAE than for those who did undergo treatment ($p < .001$).

Cholescintigraphy was performed in 16 of the 54 patients during days 9–19 after admission. Four patients showed evidence of a biloma. One patient was treated by percutaneous drainage, and the remaining three patients were treated successfully with conservative management. In all 16 patients, the localized low-density areas in the injured liver were not evident on CT images obtained at 3–6 months.

Angiography was repeated in six patients during days 7–21 after admission. Four of these patients had a biloma revealed by cholescintigraphy. In three of these four patients, extravasation of contrast medium extending within the hepatic parenchyma (pseudoaneurysms) was seen on celiac angiograms, and the three patients underwent a second TAE. Two of these three patients developed severe hypotension caused by rehemorrhage from the injured liver (on day 15 [Figs. 2G– 2I] and on day 21 [13] after admission). They immediately underwent a second TAE, which resulted in return of hemodynamic stability. In the remaining two of six patients, a second angiogram showed no extravasation of contrast medium (group A or B). All six patients recovered uneventfully and were discharged in good condition between days 26 and 97 after admission.

TABLE 3 Angiographic Findings and CT Hepatic Injury Severity Grade in 28 Patients

Angiographic Findings	CT Grade	
	3	4
A	5	0
B	8	0
C	4	3
D	3	5

Note.—Angiographic findings: A = a variable degree of avascularity and irregularity in the accumulation of contrast medium in the hepatic parenchyma or displacement of hepatic arterial branches, B = hepatic artery–portal vein fistula or disruption of hepatic artery branches, C = extravasation of contrast medium extending within the hepatic parenchyma, D = extravasation of contrast medium beyond the hepatic parenchyma.

Nonsurgical management of hepatic injury was successful in 100% of 54 patients enrolled in the protocol, and the mortality rate was 0%. The follow-up time for the patients in this study was 1–8 months (2.51 ± 1.81 months). The length of stay for all patients enrolled in the protocol was 2–129 days (22.4 ± 26.0 days).

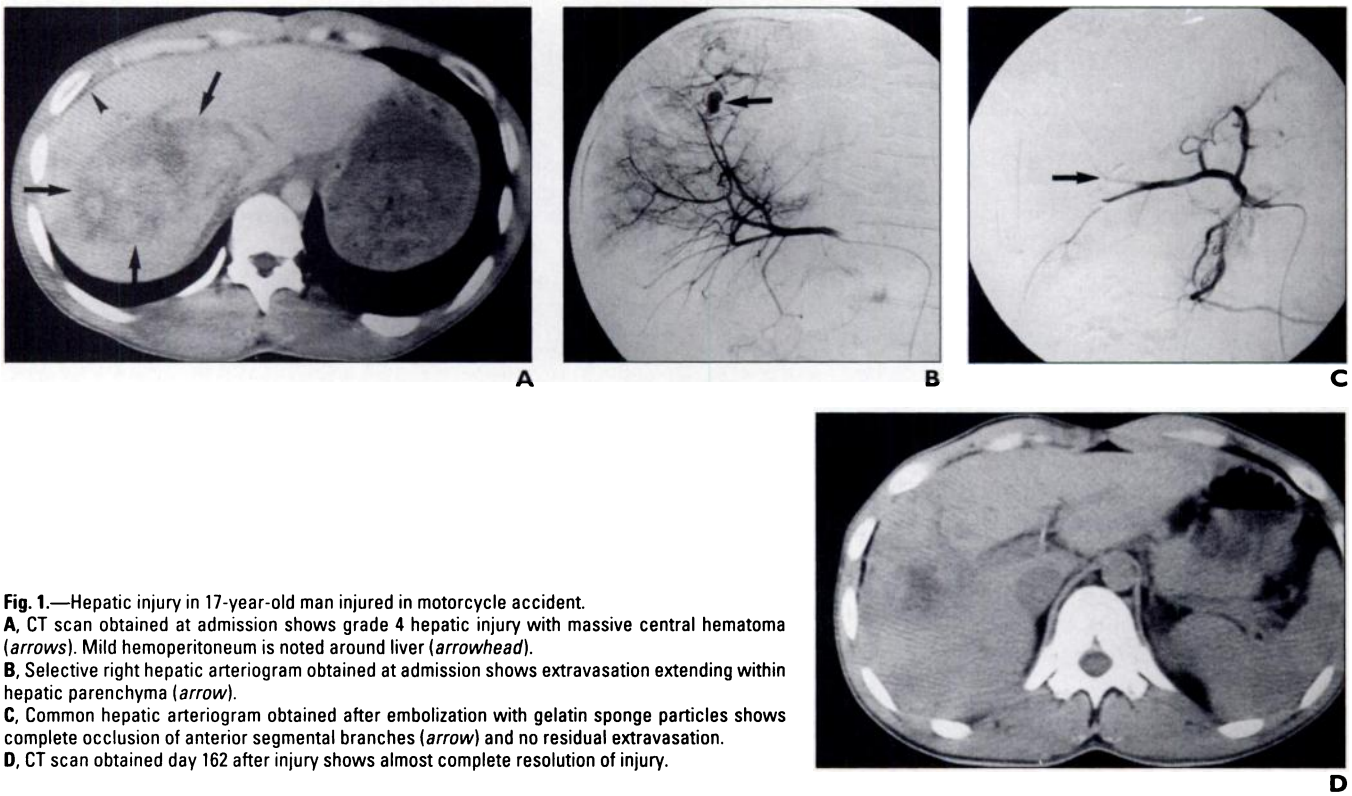

Fig. 1.—Hepatic injury in 17-year-old man injured in motorcycle accident.
A, CT scan obtained at admission shows grade 4 hepatic injury with massive central hematoma (*arrows*). Mild hemoperitoneum is noted around liver (*arrowhead*).
B, Selective right hepatic arteriogram obtained at admission shows extravasation extending within hepatic parenchyma (*arrow*).
C, Common hepatic arteriogram obtained after embolization with gelatin sponge particles shows complete occlusion of anterior segmental branches (*arrow*) and no residual extravasation.
D, CT scan obtained day 162 after injury shows almost complete resolution of injury.

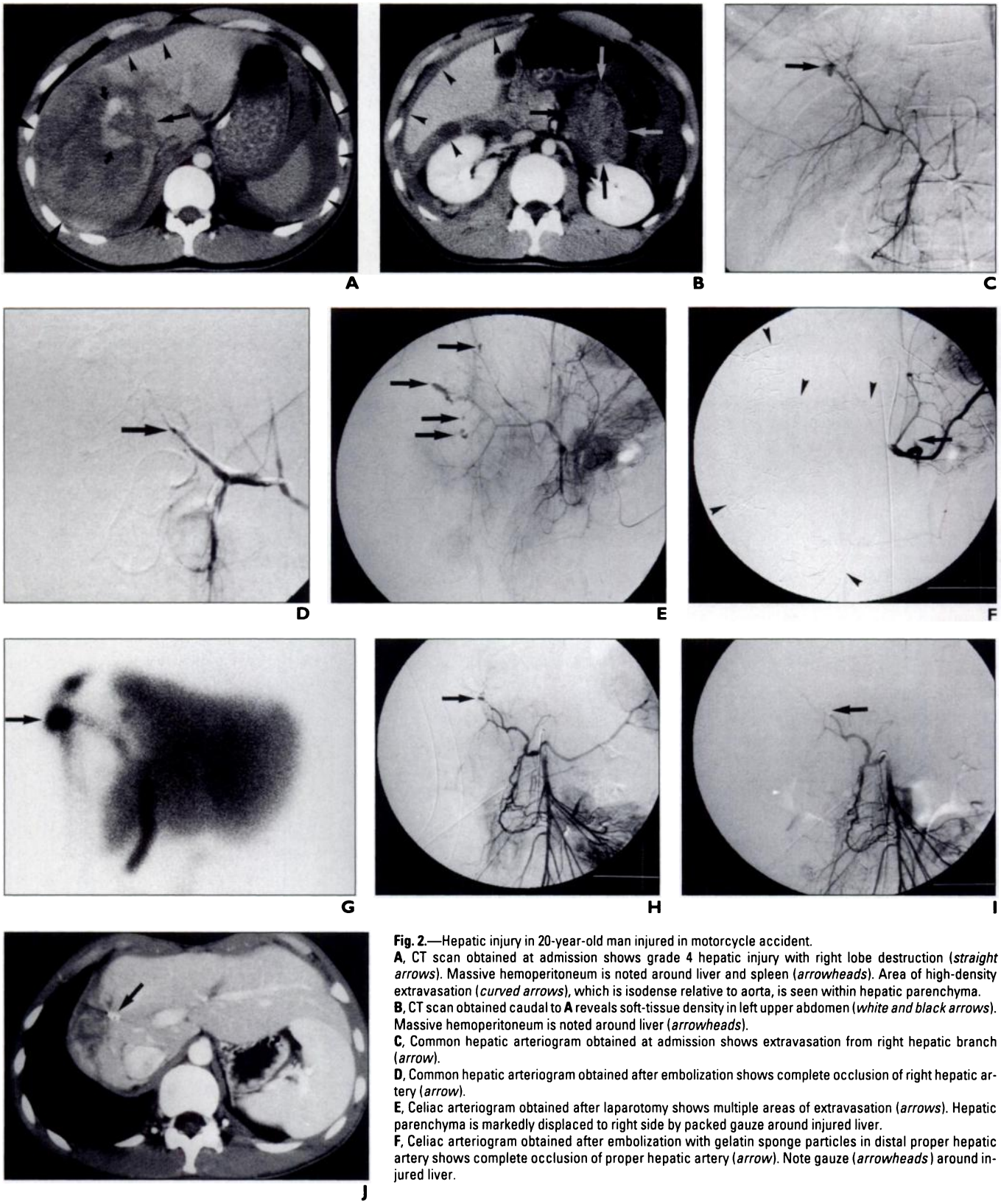

Fig. 2.—Hepatic injury in 20-year-old man injured in motorcycle accident.

A, CT scan obtained at admission shows grade 4 hepatic injury with right lobe destruction (*straight arrows*). Massive hemoperitoneum is noted around liver and spleen (*arrowheads*). Area of high-density extravasation (*curved arrows*), which is isodense relative to aorta, is seen within hepatic parenchyma.

B, CT scan obtained caudal to **A** reveals soft-tissue density in left upper abdomen (*white and black arrows*). Massive hemoperitoneum is noted around liver (*arrowheads*).

C, Common hepatic arteriogram obtained at admission shows extravasation from right hepatic branch (*arrow*).

D, Common hepatic arteriogram obtained after embolization shows complete occlusion of right hepatic artery (*arrow*).

E, Celiac arteriogram obtained after laparotomy shows multiple areas of extravasation (*arrows*). Hepatic parenchyma is markedly displaced to right side by packed gauze around injured liver.

F, Celiac arteriogram obtained after embolization with gelatin sponge particles in distal proper hepatic artery shows complete occlusion of proper hepatic artery (*arrow*). Note gauze (*arrowheads*) around injured liver.

G, Cholescintigram obtained day 9 after injury shows biloma (*arrow*) in lateral side of right lobe.

H, Superior mesenteric arteriogram obtained day 15 after injury shows extravasation extending within hepatic parenchyma from right hepatic branch (*arrow*), which corresponds to area of biloma seen on cholescintigraphy.

I, Superior mesenteric arteriogram obtained after embolization shows complete occlusion of right hepatic branch (*arrow*). Because of occlusion of celiac trunk, microcatheter is inserted to right hepatic branch from inferior pancreaticoduodenal artery, posterior pancreaticoduodenal artery, and gastroduodenal artery. Stainless-steel microcoils were deployed in right hepatic branch through microcatheter.

J, CT scan obtained on day 182 after injury shows almost complete resolution of injury. Small high-density area (*arrow*) represents deployed microcoils.

Twelve patients remained in the hospital for more than 40 days. Five of these 12 patients required treatment for associated injuries, most often in rehabilitation units (cerebral contusion with unconsciousness [$n = 1$], severe pelvic fracture [$n = 3$], or cervical spine fracture [$n = 1$]). For the remaining 49 patients, the length of stay was 2–97 days (18.0 ± 16.0 days).

TABLE 4 Shock Index and Average Volume of Fluid Resuscitation in Patients with High-Grade (≥3) Blunt Hepatic Injury

TAE	Shock Index (beats per min/mm of Hg)[a]		Average Volume of Fluid Resuscitation (ml/kg per hr)	
None ($n = 13$)	3 hr after admiss.	12 hr after admiss.	3 hr after admiss.	24 hr after admiss.
	0.77 ± 0.16	0.73 ± 0.17	6.84 ± 4.83[b]	2.95 ± 1.36
TAE ($n = 15$)	Immediately before	1 hr after TAE	Admiss. until TAE	24 hr after TAE
	1.25 ± 0.23[c]	0.76 ± 0.12[c]	19.93 ± 10.91[b,d]	2.78 ± 0.87[d]

Note.—TAE = transcatheter arterial embolization, Admiss. = admission. Data are expressed as mean ± SD.
[a]Shock index = heart rate (beats per min)/systolic blood pressure (mm of Hg).
[b-d]$p < .001$ for paired Student's t tests.

Discussion

Nonsurgical management of blunt hepatic injuries in pediatric patients who are hemodynamically stable and who have been resuscitated rapidly has been extensively documented [2–4, 7]. CT of the abdomen and pelvis has been shown to be a useful means of guiding nonsurgical management in these patients [5, 6]. Strict adherence to the CT criteria has resulted in a success rate of 80–100%. However, nonsurgical management of blunt hepatic injury in adult patients, especially those with severe complex hepatic injury, remains controversial. Many trauma surgeons who deal with adult hepatic injuries advocate the time-tested approach of early operative intervention to keep blood transfusion to a minimum and to avoid the possibility of overlooking associated intraabdominal injuries [14, 15]. However, several researchers have reported that bleeding from 50–86% of all hepatic injuries has already stopped at the time of laparotomy [16, 17]; this finding has resulted in a trend toward the use of CT imaging and nonsurgical management of blunt hepatic injuries in hemodynamically stable patients. Since 1988, this treatment approach at several level I trauma centers has resulted in a success rate of more than 84% [2–4, 7]. However, the percentage of patients with blunt hepatic injuries who could be managed nonoperatively varied widely, ranging from 16% to 72% [16]. Other studies have shown that at least 20–45% of all patients with blunt hepatic injuries could be treated nonoperatively [2, 6, 14, 15], confirming this wide variability. CT appears to reliably predict the outcome of mild hepatic injury (grade 1 or 2), and patients with such injuries can generally be managed nonoperatively. However, patients with severe complex hepatic injury (grade 3–5) will generally require surgery. We favor the view that CT does not provide sufficient vascular information to be useful for evaluating hemodynamics in these latter patients.

TAE of blunt hepatic injury was first recognized as a safe and effective treatment for the control of recurrent postoperative hemorrhage, hemobilia, and hepatic artery–portal vein fistulas in the late 1970s [18–22]. Hashimoto et al. [10] also showed the efficacy of emergency TAE in four patients with severe complex hepatic injury and suggested that this method may be useful in nonsurgical management of unstable patients with severe hepatic injury.

We proposed that detailed examination of the vasculature in patients with severe complex hepatic injury using angiography and appropriate angiographic intervention could have therapeutic potential. To achieve these objectives, we reorganized the staff and facilities of our trauma and critical care center so that both surgeons and radiologists are available at all times to examine and treat trauma victims. This reorganization also required the construction of new CT, angiography, and surgery suites close to the emergency department.

In our series, nonsurgical management of severe complex hepatic injury (grade 3 or 4) using angiography and TAE was successful in all 28 patients. Furthermore, 90% (54/60) of all patients with blunt hepatic injury were treated nonoperatively. We believe that this high success rate is the result of detailed angiographic examination and appropriate use of TAE.

In our study, patients who underwent TAE required a significantly larger volume of fluid for resuscitation than did those who did not undergo this procedure. The shock index and the volume of fluid required for resuscitation before TAE were higher than the values noted after this procedure. TAE reduced these values to levels similar to those of the group that did not undergo this procedure (Table 4), indicating that our patients who required TAE had a greater degree of hemodynamic instability than did those not requiring this procedure. Therefore, TAE also appears to be an appropriate therapy for patients who have hepatic injury and in whom a massive volume of fluid resuscitation is required to maintain hemodynamics.

We did not perform angiography for patients with mild hepatic injury (grade 1 or 2). Many researchers have reported that these patients could be treated using conservative management alone and that these mild injuries are not accompanied by major vascular injury, so that the associated bleeding would halt spontaneously [2–7]. In our series, patients with grade 1 or 2 injuries were hemodynamically stable initially or after modest fluid resuscitation, and CT scans showed stable or improving injuries. Our results confirm that patients with grade 1 or 2 hepatic injuries can be treated sufficiently with conservative management and do not require vascular examination by angiography.

TAE in patients with blunt hepatic injury has not been proven to be effective for venous or bile duct injury. Bleeding caused by portal vein or hepatic vein injury will continue after TAE. However, bleeding from these veins will often halt spontaneously because of intraabdominal tamponade. In our patient (Fig. 2), TAE was successful, and the arterial bleeding stopped. However, the subsequent laparotomy caused additional blood loss by releasing the intraabdominal pressure. Furthermore, the removal of clots on the injured liver caused catastrophic shock from uncontrollable bleeding. TAE was repeated immediately after laparotomy, which again halted the arterial bleeding and improved the patient's hemodynamic state. This case indicates that release of intraabdominal pressure by laparotomy causes additional blood loss from the injured artery and vein and illustrates an important advantage of TAE over surgery.

Bilomas developed in four of the 54 patients in our series. This incidence is similar to that reported by others [23]. However, in three of these four patients, pseudoaneurysms, which were identical to the areas of biloma seen on cholescintigraphy, were identified on the second angiogram. We proposed the following mechanism for this finding: the presence of bile delays healing of a liver wound [24], inflammatory reaction to extravasated bile injures nearby vessels, and, ultimately, this process could lead to rupture of blood vessels. In two of

these three patients, delayed hemorrhage was caused by pseudoaneurysms that were presumed to have been induced by bile leakage. Delayed hemorrhage is a life-threatening complication of hepatic injury that demands rapid treatment. Therefore, an assessment of bile duct injury should be performed after TAE in patients with severe complex hepatic injury. If a biloma is detected, angiography should be performed to assess vascular injury that may result in delayed hemorrhage. Furthermore, TAE for delayed hemorrhage seems to be an effective therapy.

The length of stay in the hospital for our patients was not excessive. The mean number of days spent in the hospital was similar to that of patients who underwent nonsurgical treatment in multiinstitution studies [4, 7]. Several authors have reported that the mean length of hospitalization for patients managed operatively was either comparable or exceeded that of patients managed nonoperatively [2, 16].

The treatment protocol used in this study defined a framework for the use of TAE. In addition, the protocol included criteria for emergency management. Two of our patients required emergency surgery. We believe that some patients will always require emergency laparotomy. However, hepatic trauma in patients without associated injuries or catastrophic shock or both can usually be managed without surgery by this protocol. Our results should encourage more extensive evaluation of angiography and embolization for patients with hepatic injury as an alternative to surgery.

References

1. Civil ID, Schwab CW. The abbreviated injury scale, 1985 revision: a condensed chart for clinical use. *J Trauma* **1988**;28:87–90
2. Farnell MB, Spencer MP, Thompson E, Williams HJ, Much P, Ilstrup DM. Nonoperative management of blunt hepatic trauma in adults. *Surgery* **1988**;104:748–756
3. Pachter HL, Spencer FC, Hofstetter SR, Liang HG, Coppa GF. Significant trends in the treatment of hepatic trauma: experience with 411 injuries. *Ann Surg* **1992**;215:492–502
4. Pachter HL, Knudson MM, Esrig B, et al. Status of nonoperative management of blunt hepatic injuries in 1995: a multicenter experience with 404 patients. *J Trauma* **1996**;40:31–38
5. Foley WD, Ctes JD, Kellman GM, et al. Treatment of blunt hepatic injuries: role of CT. *Radiology* **1987**;164:635–638
6. Mirvis SE, Whitley NO, Vainwright JR, Gens DR. Blunt hepatic trauma in adults: CT-based classification and correlation with prognosis and treatment. *Radiology* **1989**;171:27–32
7. Pachter HL, Hofstetter SR. The current status of nonoperative management of adult blunt hepatic injuries. *Am J Surg* **1995**;169:442–454
8. Rubin BE, Katzen BT. Selective hepatic artery embolization to control massive hepatic hemorrhage after trauma. *AJR* **1977**;129:253–256
9. Sclafani SJR. Angiographic control of intraperitoneal hemorrhage caused by injuries to the liver and spleen. *Semin Intervent Radiol* **1985**;2:139–147
10. Hashimoto S, Hiramatsu K, Ido K, Yosii H, et al. Expanding role of emergency embolization in the management of severe blunt hepatic trauma. *Cardiovasc Intervent Radiol* **1990**;13:193–199
11. Esenten M, Ralls PW, Colletti P, et al. Posttraumatic intrahepatic biloma: sonographic diagnosis. *AJR* **1983**;140:303–305
12. Baker SP, O'Neill B, Haddon W Jr, Long WB. The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. *J Trauma* **1974**;14:187–196
13. Hagiwara A, Yukioka T, Shimazaki S, Megawa T, Matuda H. Delayed hemorrhage following transcatheter arterial embolization for blunt hepatic injury. *Cardiovasc Intervent Radiol* **1993**;16:380–383
14. Hiatt JR, Harrier HD, Koening BV, Ransom KJ. Nonoperative management of major blunt injury with hemoperitoneum. *Arch Surg* **1990**;125:101–103
15. Federico JA, Horner WR, Clark DE, Isler RJ. Blunt hepatic trauma: nonoperative management in adults. *Arch Surg* **1990**;125:905–909
16. Meredith JW, Young JS, Bowling J, et al. Nonoperative management of blunt hepatic trauma: the exception or the rule. *J Trauma* **1994**;36:529–535
17. Sugimoto K, Asari Y, Sakaguchi T, et al. Endoscopic retrograde cholangiography in the nonsurgical management of blunt liver injury. *J Trauma* **1993**;35:192–199
18. Knudson MM, Lim RC, Oakes DD, Jeffrey RB Jr. Nonoperative management of blunt liver injuries in adults: the need for continued surveillance. *J Trauma* **1990**;30:1494–1550
19. Jander HP, Laws HL, Kogutt MS, Mihas AA. Emergency embolization in blunt hepatic trauma. *AJR* **1977**;129:249–252
20. Bass EM, Crosier JH. Percutaneous control of post-traumatic hepatic hemorrhage by Gelfoam embolization. *J Trauma* **1977**;17:61–63
21. Walter JF, Paaso BT, Cannon WB. Successful transcatheter embolic control of massive hematobilia secondary to liver biopsy. *AJR* **1976**;127: 847–849
22. Sclafani SJR, Nayaranaswamy T, Mitchell WG. Radiologic management of traumatic hepatic artery-portal vein fistulae. *J Trauma* **1981**;21:576–580
23. Mirvis SE, Dunham CM. Abdomen/pelvic trauma. In Mirvis SE, Young JWR, eds. *Imaging in trauma and critical care*, 1st ed. Baltimore: Williams & Wilkins, **1992**:178–180
24. Sandblom P, Mirkovitch V, Gardiol D. The healing of liver wounds. *Ann Surg* **1976**;183:679–684