Emergency Embolization in Blunt Hepatic Trauma

H. PETER JANDER,[1] HENRY L. LAWS,[2] MARVIN S. KOGUTT,[1] AND ANASTASIOS A. MIHAS[3]

The right hepatic artery in a patient with traumatic liver tear was embolized with Gelfoam to stop an otherwise uncontrollable hemorrhage. The procedure was probably life saving in this patient. Transient elevated transaminase and bilirubin levels returned to normal 3 weeks after embolization. A repeat hepatic arteriogram 4 months after embolization demonstrated good revascularization of the right hepatic lobe from the left hepatic, left gastric, and gastroduodenal arteries. This case demonstrates the therapeutic value and safety of selective hepatic arterial branch embolization in massive life-threatening hemorrhage.

Control of hemorrhage from severely lacerated liver parenchyma constitutes a formidable clinical problem. Surgical techniques, even though well established, are usually drastic and often fruitless [1-3]. Furthermore, in cases of severe and multiple associated trauma the patient may be a poor surgical risk. Selective arterial embolization in patients with massive hemorrhage has been successfully used in pelvic trauma [4, 5], gastrointestinal lesions [6, 7], renal lacerations [8, 9], the head and neck [10], and cancer [11]. Experience with hepatic embolization so far has been limited to metastatic disease [12], complications of hepatic biopsy [13], and experiments in dogs [14]. We report the first case of successful control of massive hepatic hemorrhage due to blunt trauma by embolization in a patient who was a poor surgical risk.

Case Report

An 18-year-old white female was admitted in profound hemodynamic shock following an automobile accident. She had multiple fractures of facial bones, pelvis, and both lower extremities. Peritoneal lavage revealed gross blood. At laparotomy a lacerated spleen was resected; a small amount of intraperitoneal hemorrhage, a moderately small retroperitoneal hemorrhage at the pelvic brim (not considered to be expanding), and a tiny (1 cm) subcapsular hematoma over the right dome of the liver were found. The liver and portal system otherwise appeared intact. Laparotomy was followed by nasal and oral packing to control hemorrhage, tracheostomy, internal fixation of the left femur and tibia with Steinman pins, and casting of the right leg.

Since the patient continued to require blood transfusions to maintain adequate blood pressure, it was felt that hemorrhage was continuing. Selective arteriography revealed two bleeding sties: one in the distribution of the obturator branch of the right internal iliac artery, and the other from a peripheral branch of the right hepatic artery resulting in a large subcapsular hepatic hematoma (figs. 1*B* and 1*C*). The right hepatic artery originated from the superior mesenteric artery (fig. 1*A*). Further surgery was considered an unacceptable approach to the bleeding because of the patient's poor condition.

Both bleeding arteries were embolized under fluoroscopic control with Gelfoam shavings suspended in 76% meglumine sodium diatrizoate (Renografin 76) injected through selectively placed no. 7 French polyethylene endhole catheters. Complete control of hemorrhage required about 2-3 ml of Gelfoam shaving suspension at each site and was verified by control angiography (fig. 2). Thereafter, the patient dramatically stabilized, requiring only two more units of blood that day. She had required a total of 49 units of blood during the preceding 32 hr.

There was a rapid and constant rise of platelets and hemoglobin to normal levels over the next 9 days after embolization. SGOT, lactic dehydrogenase, and total bilirubin rose to peak levels of 16-32 times normal within 1-11 days and fell with an initial sharp and a secondary more gradual drop, reaching near normal levels about 3-5 weeks after embolization. The alkaline phosphatase reached a level of 2.5 times normal 16 days after embolization. A return to normal levels was not observed during the first month after embolization.

For the initial 10 days of hospitalization, the extensive injuries and subsequent pulmonary insufficiency necessitated maintenance on controlled ventilation using an FIO_2 of 40%-80% to maintain an arterial pO_2 of 80-150 mm Hg. Positive end-expiratory pressure up to 15 cm of water and intermittent mandatory ventilation were used.

The patient made a slow but progressive recovery. Repeat hepatic arteriography 4 months after emoblization revealed total occlusion of the right hepatic artery 1 cm distal from its origin from the superior mesenteric artery (fig. 3*A*). The right hepatic lobe was completely revascularized through intrahepatic translobar anastomoses originating from an enlarged left hepatic artery and through anastomotic channels from the gastroduodenal and left gastric artery (fig. 3*B*). In addition, some revascularization appeared to have taken place through the inferior phrenic artery originating from the superior mesenteric artery (fig. 3*A*). The previously embolized right obturator artery had reestablished its circulation through the enlarged tortuous uterine artery and several small anastomotic branches.

Discussion

For many years major arterial branches of the liver have been considered functional end arteries [15-17]. More recent clinical experience demonstrated the usefulness of ligation or excision of hepatic arteries in controlling exsanguinating hemorrhage [3] and revealed the surprisingly rapid revascularization of the human liver through both preexisting and newly formed collaterals after arterial interruption [18-20]. In our case the right hepatic lobe was completely revascularized from translobar, left gastric, gastroduodenal, and inferior phrenic collaterals 4 months after embolization. These findings and those of others [13, 18, 19] demonstrate that indeed the hepatic artery is not an end artery. Since the liver survives transient ischemia by increased extraction of oxygen from the portal blood [21], functional integrity of

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[1] Department of Radiology, University of Alabama Medical Center, 619 S. Nineteenth Street, Birmingham, Alabama 35233. Address reprint requests to H. P. Jander.

[2] Department of Surgery, University of Alabama Medical Center, Birmingham, Alabama 35233.

[3] Department of Gastroenterology, University of Alabama Medical Center, Birmingham, Alabama 35233.

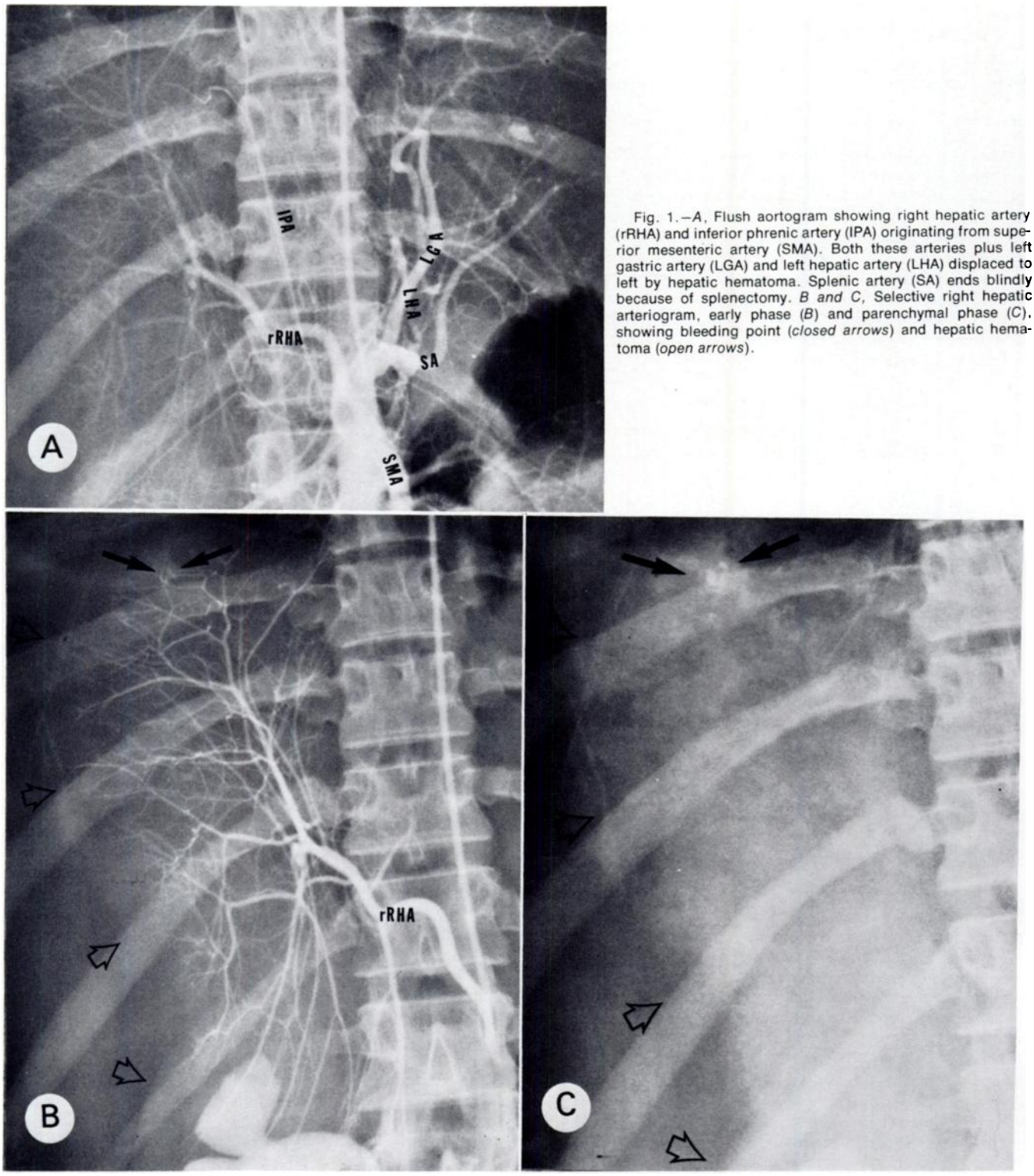

Fig. 1.—*A*, Flush aortogram showing right hepatic artery (rRHA) and inferior phrenic artery (IPA) originating from superior mesenteric artery (SMA). Both these arteries plus left gastric artery (LGA) and left hepatic artery (LHA) displaced to left by hepatic hematoma. Splenic artery (SA) ends blindly because of splenectomy. *B and C*, Selective right hepatic arteriogram, early phase (*B*) and parenchymal phase (*C*), showing bleeding point (*closed arrows*) and hepatic hematoma (*open arrows*).

the portal system, including unimpeded mesenteric arterial influx, is a prerequisite to hepatic arterial embolization. Both fasting and supplemental oxygen increase portal vein oxygenation [22].

Since the liver responds to hypoxemia with mobilization of glucose deposits which may eventually lead to glucose depletion, close monitoring for hypoglycemia is necessary. Infusion of hypertonic glucose prevents glucose mobilization and increases hepatic blood flow [23]. Gallbladder infarction following hepatic artery embolism has recently been reported [24], but several large surgical series indicate that prophylactic cholecystectomy is rarely necessary [3, 25–27].

Even though transaminase, alkaline phosphatase, and total bilirubin levels are of limited quantitative value as indicators of hepatic damage in an extensively trauma-

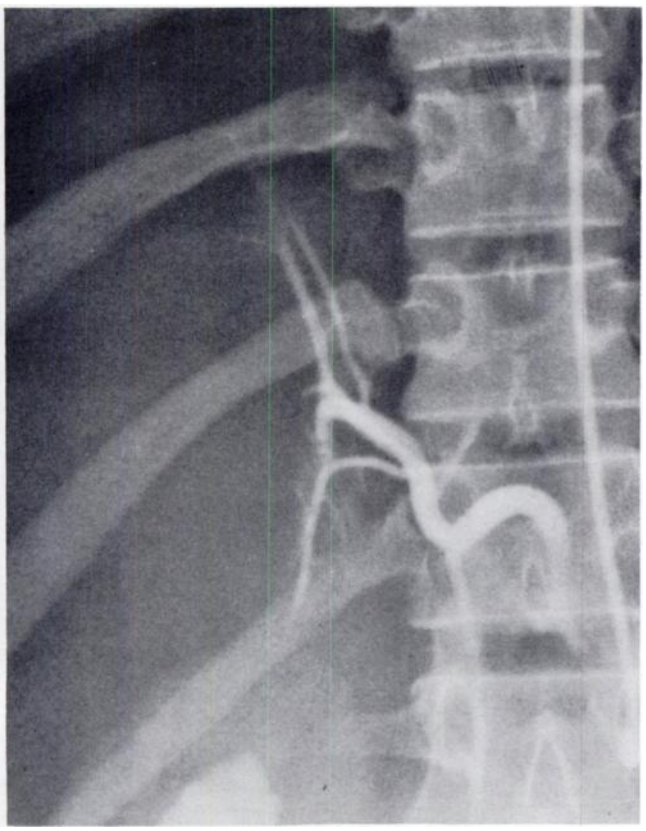

Fig. 2.—Arteriogram immediately after embolization of right hepatic artery showing complete control of hemorrhage.

tized patient, they demonstrated essentially complete functional recovery of the liver in our patient by no later, and possibly sooner, than 4 weeks after embolization.

This case furthermore revealed the limitation of exploratory laparotomy in evaluating hepatic and pelvic bleeding sites since only a small subcapsular hepatic and a pelvic hematoma were found, and they were considered insignificant. Selective arteriography revealed the serious nature of these bleeding sites. Hepatic and internal iliac arterial embolization was life saving in this patient. This interventional radiologic procedure is an attractive and possibly preferable alternative to surgery.

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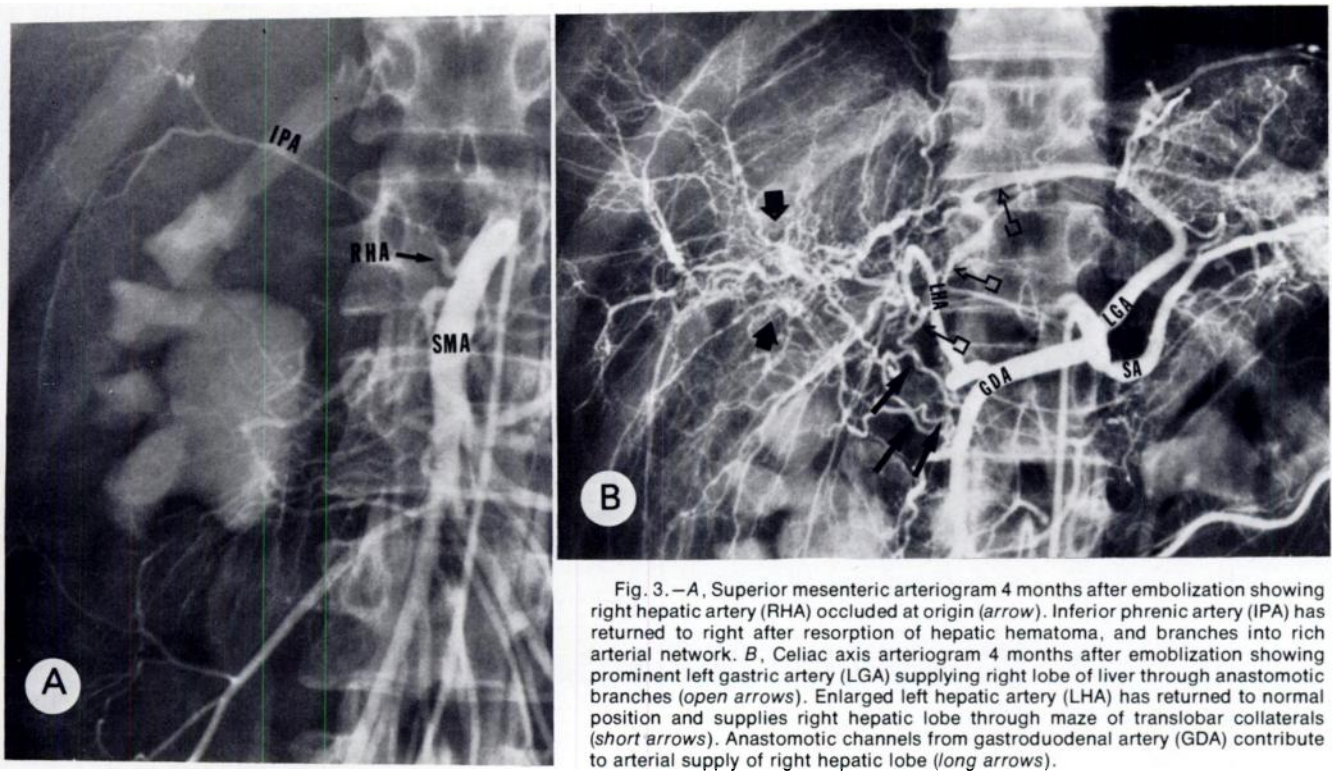

Fig. 3.—*A*, Superior mesenteric arteriogram 4 months after embolization showing right hepatic artery (RHA) occluded at origin (*arrow*). Inferior phrenic artery (IPA) has returned to right after resorption of hepatic hematoma, and branches into rich arterial network. *B*, Celiac axis arteriogram 4 months after emoblization showing prominent left gastric artery (LGA) supplying right lobe of liver through anastomotic branches (*open arrows*). Enlarged left hepatic artery (LHA) has returned to normal position and supplies right hepatic lobe through maze of translobar collaterals (*short arrows*). Anastomotic channels from gastroduodenal artery (GDA) contribute to arterial supply of right hepatic lobe (*long arrows*).

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