Journal of Surgical Oncology 29:129–131 (1985)

Spontaneous Rupture of Hepatocellular Carcinoma

SAMUEL B. VAN LANDINGHAM, MD, JOHN C. HENDRICKS, MD, AND JOHN W. ROBERTS, MD,
From the Department of Surgery, Scott and White Clinic, Scott and White Memorial Hospital, Scott, Sherwood and Brindley Foundation, Texas A&M University College of Medicine, Temple, Texas

Spontaneous rupture of a primary hepatocellular carcinoma is an extremely rare event in the western hemisphere. Only a handful of single-case reports have been authored in the continental United States. A ruptured hepatoma carries a dismal prognosis and is usually beyond a "resection for cure" stage. In this report, two cases of spontaneously ruptured primary hepatocellular carcinoma are described. Both cases involved cirrhotic livers, and the tumor in each case was resected to attempt cure. One patient survived nearly 2 years; the other is alive and well at this time.

KEY WORDS: **rupture, hepatoma, cirrhosis**

INTRODUCTION

Primary hepatocellular carcinoma is uncommon in the United States, and spontaneous rupture is even more rare. Spector and Chodoff reviewed the world literature in 1950 and found only 14 cases of spontaneous rupture of liver malignancies [1]. Primary liver carcinoma is much more prevalent in Asia and Africa, and since 1950, several series of ruptured liver cancer have been authored there. The prognosis is usually poor, and in a recent report of 63 cases from Thailand, the largest to date, only a single case was found to be surgically resectable. This patient did not survive 4 months [2].

In the span of 2 years, two cases of spontaneously ruptured hepatocellular carcinoma have been diagnosed and treated at the Scott and White Clinic. Both cases presented as acute abdominal crises mandating surgical exploration. Both cases involved cirrhotic livers; and in both instances, resections were performed to attempt cure.

CASE REPORTS

Case Report A

A 65-year-old male was admitted to Scott and White Clinic and Hosptial on February 20, 1982, complaining of abdominal pain associated with nausea and vomiting. Physical examination revealed an obese abdomen which was mildly tender; bowel sounds were active. Initial hemoglobin was 11.8 g/dl. This fell to 9.3 g/dl on serial observation. No source of GI blood loss could be identified. CT scan demonstrated a soft tissue mass adjacent to the caudate lobe of the liver. At celiotomy a 3 × 4 cm neocrotic, bleeding subcapsular tumor was identified in the lateral segment of the left lobe of a cirrhotic liver. Bleeding was controlled with suture ligatures, and a large amount of blood was removed from the peritoneal cavity; biopsy of the mass suggested metastic tumor, but permanent sections subsequently disclosed primary hepatocellular carcinoma and cirrhosis from chronic active hepatitis. The patient recuperated from surgery and underwent celiac angiography on April 1, 1982. Since no tumor was identified by angiography, the patient was explored on April 2, 1982, and a left lateral segmentectomy was performed using the Lin hepatic clamp and "crush" technique. A 3-cm nodule in the surgical specimen suggested primary hepatocellular carcinoma on mi-

Accepted for publication June 11, 1984.

Address reprint requests to Samuel B. Van Landingham, MD, Scott and White Memorial Hospital and Clinic, 2401 South 31st Street, Temple, TX 76508.

croscopic study. The patient was discharged on April 12, 1982, and followed in surgical outpatient clinic. Elevated liver function studies led to a CT scan on April 14, 1983, which demonstrated a 7-cm mass on the anterior surface of the right lobe of the liver. A CT-directed percutaneous biopsy of the mass demonstrated grade I hepatocellular carcinoma. The patient was reexplored on March 8, 1983, and two regenerating nodules in the right lobe of the cirrhotic liver were locally excised; both contained low-grade hepatic carcinoma. Postoperative recovery was swift. The patient continued to be followed in outpatient clinic and was last seen November 16, 1983. At that time a large upper abdominal mass was palpable which was presumed to be recurrent hepatoma. The patient demonstrated signs and symptoms of congestive heart failure which were treated with diuretics and digitalis. Tumor Registry was notified of the patient's death at home on January 11, 1984.

Case Report B

A 52-year-old male was admitted to Scott and White Clinic on April 18, 1984, complaining of vague left upper abdominal discomfort associated with nausea, vomiting, and diarrhea. The "tightness" in the left abdomen was worsened by deep inspiration. The patient was syncopal upon presentation, the initial blood pressure was 74/40 mm Hg, and the pulse was 76 beats/min. Significant past medical history included surgical treatment of a perforated duodenal ulcer in 1960, and partial gastrectomy with Billroth II gastrojejunostomy for ulcer disease in 1961. The patient's wife reported that he "turned yellow" and was quite weak for several weeks following his second operation. He had received blood transfusion during that hospitalization. Physical examination revealed only mild abdominal tenderness, no rebound tenderness, and active bowel sounds. The patient's blood pressure responded promptly to infusion of blood and crystalloid. The intitial Hgb of 12.7 g/dl fell to 8.9 g/dl with serial observation. No source of blood loss through the GI tract could be identified. CT scan demonstrated a soft tissue mass in the parahepatic, paragastric region, consistent with hematoma. At celiotomy, a large amount of blood clot was removed from the peritoneal cavity. Adhesions from previous surgery had partitioned the bleeding and kept blood away from the anterior abdominal wall, probably accounting for the paucity of physical findings on initial examination.

The source of the bleeding appeared to be a 4 × 4 cm ruptured subcapsular cyst, inflammatory in appearance, on the inferior surface of the lateral segment of the left lobe of a markedly cirrhotic liver. This was excised and sent for pathologic study. Frozen section suggested it to be a poorly differentiated carcinoma, type unknown. Abdominal exploration revealed no other evidence of tumor. Wedge and needle biopsies of the liver were performed and subsequently confirmed the presence of cirrhosis. The patient recovered swiftly from surgery, but did develop significant ascites. Microscopic study of the malignant cyst demonstrated primary hepatocellular carcinoma in the permanent pathologic sections. Celiac angiography on April 16, 1984, demonstrated neovascularity in the left lateral segment of the liver, but the right lobe was free of tumor. Left lateral segmentectomy was performed the following day using the Lin hepatic clamp and "crush" technique. Blood loss was less than 150 cc. Study of the surgical specimen demonstrated only a thin rim of neoplasia along the cavity where the ruptured cyst had been excised. The margin of the resected liver was free of tumor. During the postoperative period, large volumes of serous fluid drained around a single, closed suction drain which had been placed near the raw surface of the remaining liver tissue. The drain was removed promptly, and the drain site was closed with running suture to prevent ascitic fistula.Thoracentesis and paracentesis were required postoperatively to alleviate respiratory difficulty. Sodium and fluid restriction and oral diuretics were used to control ascites. Tube feedings were utilized to provide adequate protein and caloric intake. Although liver function tests (SGOT, LDH) and total bilirubin were transiently elevated in the postoperative period, these were within normal limits at the time of the patient's discharge on April 28, 1984.

DISCUSSION

Most series concerning spontaneous rupture of primary liver cancer from Asia or Africa place the incidence at 8–14.5% [3–5]. Reports from the continental U.S. are limited to individual case reports. A series of 137 patients with primary liver cancer from Memorial Hospital in New York City made no mention of tumor rupture [6].

All reports in the literature indicate a dismal prognosis. Berman, in his exhaustive review of primary liver cancer in 1951, reported six cases of spontaneous rupture; all six patients were dead within 40 days [3]. Balasegaram [7] reported 10 cases of ruptured hepatocellular carcinoma, and of those that underwent resection, none survived more than 6 months.

A few long-term survivors have been reported. Roe [8] reported a 38-year-old noncirrhotic Chinese man who was free of disease 2 years after resection of a ruptured hepatocellular carcinoma. Ong and Taw [9] reported 42 cases of ruptured hepatoma in Hong Kong with only a single long-term survivor (alive at 5 years). Hermann and David [10] reported a 29-year-old noncirrhotic with ruptured liver cancer alive and well 9 months after resection. Inouye and Whelan [11], in a review of 205 cases of primary liver cancer in Hawaii, reported 13 cases of acute rupture. One patient was an 8-year survivor follow-

ing lobectomy, but the average survival for the 12 others was only 2.5 months.

Ong suggests the prognosis of this disease may rest on whether or not cirrhosis is present. If the liver is markedly cirrhotic, then survival, either immediately after resection or on a long-term basis, is poor. Tumors may be multicentric in cirrhosis and may recur in the hepatic remnant despite adequate resection.

While the presence of cirrhosis does not preclude curative resection, resections must be approached with caution. Hepatic reserve is limited, and regeneration is impaired in the presence of cirrhosis. Stone [12] has stated that a resection leaving only 60% of normal parenchyma is much better tolerated than supposed "minor" removal of 15% of functioning tissue from a cirrhotic liver. Lobar resections are to be avoided; resections should be limited to segmentectomies and local excisions [13].

Both of our cases involved tumors localized to the left lateral segment. The Lin hepatic clamp and "crush" technique were used to minimize blood loss. Conventional "finger fracture" technique of liver resection is nearly impossible in the hard, fibrotic liver of cirrhosis. The "crush" technique of liver resection first advocated by Lin in 1974 is very useful in this circumstance [14].

Most series of primary liver cancer report a 60–70% incidence of cirrhosis [11]. We were unaware of the presence of cirrhosis in our two patients prior to celiotomy. With evidence of hemoperitoneum in a known cirrhotic, one might suspect ruptured primary liver cancer. In this setting, we would suggest angiography preoperatively in order to localize the bleeding, determine resectability, and help the surgeon in planning a resection. It is extremely difficult to determine the extent and resectability of a hepatic tumor in a markedly cirrhotic liver at the time of surgery.

CONCLUSION

An acute abdominal crisis associated with falling hematocrit in the cirrhotic patient without obvious source of blood loss suggests the presence of ruptured hepato cellular carcinoma. Should this diagnosis be entertained preoperatively, angiography is suggested to localize the source of bleeding and determine resectability. Surgical resection is the only effective mode of therapy for hepatocellular carcinoma. Neither rupture of the tumor nor the presence of cirrhosis precludes curative resection. Hepatic resections must be approached cautiously in cirrhotics and should be limited to segments or local excisions. Use of the Lin heaptic clamp and "crush" technique is recommended.

REFERENCES

1. Specter J, Chodoff RJ: Massive intraperitoneal hemorrhage from carcinoma of the liver: Review of the literature and case report. Surgery 27:457, 1950.
2. Chearanai O, Plengvanik U, Asavanich C, et al: Spontaneous rupture of primary hepatoma: Report of 63 cases with particular reference to the pathogenesis and rationale: Treatment by hepatic artery ligation. Cancer 51:1532–1536, 1983.
3. Berman C: "Primary Carcinoma of the Liver." London: Lewts, 1951, p 27.
4. Viranewatti V, Stapanakul C: Primary carcinoma of the liver: Analysis of 90 cases. In "Proceedings of World Congress of Gastroenterology." Washington DC, 1958, pp 516–525.
5. Ong GB, Chu EPH, Yu FTK, Lee TC: Spontaneous rupture of hepatocellular carcinoma. Br J Surg 52:123–129, 1965.
6. El-Domeini AA, Huvos AG, Goldsmith HS, Foot FW: Primary malignant tumors of the liver. Cancer 27:7, 1971.
7. Balasegaram M: Rupture of liver cell carcinoma. Aust NZ J Surg 37:332–337, 1968.
8. Roe B: Massive hemorrhage from malignant hepatoma: Report of a case with subsequent transdiaphragmatic resection. Arch Surg 65:912, 1952.
9. Ong GB, Taw JL: Spontaneous rupture of hepatocellular carcinoma. Br Med J 4:146–149, 1972.
10. Hermann RE, David ED: Spontaneous rupture of the liver caused by hepatomas. Surgery 74:715–718, 1973.
11. Inouye AA, Whelan TJ Jr: Primary liver cancer: A review of 205 cases in Hawaii. Am J Surg 138:53–61, 1979.
12. Stone HH, Long WD, Smith RB III, Hayes CD: Physiologic Considerations in Major Hepatic Resections. Am J Surg 138:53–61, 1979.
13. Nyhus L (ed): Surgery Annual, 1984. Norwalk, Connecticut: Appleton-Century Crofts, 1984.
14. Lin T: A Simplified Technique for Hepatic Resection: The Crush Method. Ann Surg 180:285, 1974.