
Ruptured Primary Hepatocellular Carcinoma at Chulalongkorn University Hospital: A Retrospective Study of 32 Cases

ATTAPHOL PAWARODE, M.D.*,
NARIN VORAVUD, M.D.*

Abstract

Rupture of Primary hepatocellular carcinoma (HCC) is relatively common in high incidence areas including Thailand. There have been attempts to establish a standard treatment to manage this phenomenon. We retrospectively reviewed the records of patients with HCC from January 1989 to June 1997, and ten per cent (32/306) had tumor rupture during the course of the disease. Overall median survival of the patients with tumor rupture was 2.7 months [95% confidence interval (CI), 0-5.9 months] that was not significantly different from that of the patients without rupture (median 6.6 months; 95% CI, 4.0 - 9.1 months) ($P = 0.4605$). Among the ruptured group, the patients treated with surgical intervention survived longer than those receiving supportive care alone (median = 15.5 months; 95% CI, 8.7-22.2 months and median = 0.4 months; 95% CI, 0.2-0.5 months, $P = 0.0027$). The resectional and non-resectional surgical subgroups also had better survival than the supportive group ($P = 0.0300$ and $P = 0.0209$, respectively). In conclusion, surgical intervention, if applicable, should be performed in managing ruptured HCC.

Primary hepatocellular carcinoma (HCC) is prevalent in some parts of the world especially Far Eastern Asia and sub-Saharan Africa. The dreadful complication of tumor rupture is relatively common in these high incidence areas, occurring 7-15 per cent of the time during the course of the disease⁽¹⁻⁶⁾. The treatment of patients with this complication poses a difficult problem. Until now, there have been no standard strategies to manage this disastrous phenomenon. In this study, we

investigated the natural history of ruptured hepatocellular carcinoma and evaluated whether some modalities of treatment, especially surgical procedures, altered patient survival.

MATERIAL AND METHOD

Three hundred and six cases of HCC treated at Chulalongkorn University Hospital, Bangkok, Thailand, from January 1989 to June 1997 were retrospectively reviewed. Thirty two cases

* Division of Medical Oncology, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand.

(10.4 %) were found to have developed tumor rupture during the course of the disease. The diagnosis of HCC was established by needle biopsy in 11 patients, fine-needle aspiration in 3 cases, surgical biopsy in 9 cases. In the remaining 9 patients, the diagnosis was based on clinical features, imaging studies and serum alpha-fetoprotein (AFP) levels (≥ 400 IU/mL)(7). The diagnosis of ruptured HCC was based on clinical diagnosis and the presence of hemoperitoneum by either abdominal tapping or exploratory laparotomy.

Demographic data of patients with and without tumor rupture are summarized in Table 1. Patients' stages were classified according to the TNM(8) and Okuda's staging system(9). The data of the ruptured and unruptured groups were compared using the Pearson chi-square with Yates' correction for continuity. Patients' survival from the time of diagnosis were analyzed using the Kaplan Meier method and comparisons were made by use of the Log Rank test.

RESULTS

There were 27 males and 5 females with a male-to-female ratio of 5.4 : 1. The median age was 48.5 years (range 4.9-82.3 years). Median time of follow-up was 2.6 months (range 0.2-33.9

months). Associated cirrhosis was histologically revealed in 80 per cent (16/20).

Of 32 patients, 17 cases (53.1%) developed tumor rupture as the first clinical presentation (acute abdomen and those of hemorrhagic shock) without other previous symptoms or signs. Among this subgroup, only 2 cases had a history of blunt abdominal trauma and 8 patients underwent surgical treatment. Six patients had emergency surgery; 3 cases had tumor resection, one had hepatic artery ligation and the other two had suturing and packing of bleeding sites. Another two had delayed surgical treatments including right hepatic lobectomy and hepatic artery ligation. The remaining nine cases received supportive care for the rupture only.

Three patients (9.4%) had neither symptoms nor signs of HCC rupture but it was found incidentally during elective hepatic resections. Packing was then performed instead in one of them. One patient received emergency transcatheter arterial embolization and expired from liver failure 14 days later. Four patients had tumor rupture after treatment for HCC had been implemented (transcatheter oil chemo-embolization (TOCE), systemic chemotherapy and combined type), but no specific treatments for the rupture were carried

Table 1. Patients characteristics.

Characteristics	Ruptured group		Unruptured group	
Number	32		274	
Age, median in year (range, year)	48.5 (4.9-82.3)		52.1 (2.3-85.3)	
Male-to-female ratio	5.4 : 1		5.7 : 1	
Associated cirrhosis, n/n (%)	16/20 (80)		101/202 (50)	
Staging				
Okuda				
I, n (%)	6	(18.8)	31	(11.3)
II, n (%)	21	(65.5)	189	(69.0)
III, n (%)	5	(15.6)	54	(19.7)
TNM				
I, n (%)	0	(0)	3	(1.1)
II, n (%)	8	(25.0)	71	(25.9)
III, n (%)	5	(15.6)	35	(12.8)
IVA, n (%)	12	(37.5)	132	(48.2)
IVB, n (%)	7	(21.9)	33	(12.0)
Treatment received				
Surgical Intervention				
Non-resectional type, n (%)	5	(15.6)	0	(0)
Resectional type, n (%)	7	(21.9)	42	(15.3)
Supportive care, n (%)	20	(62.5)	232	(84.7)

Table 2. Characteristics of patients with ruptured HCC

Characteristics	Details	
Patient status		
Dead, n (%)	24	(75.0)
Alive with disease, n (%)	4	(12.5)
Lost to follow-up, n (%)	4	(12.5)
Cause of death		
Ruptured hepatocellular carcinoma, n (%)	9	(37.5)
Liver failure, n (%)	5	(20.8)
Upper GI hemorrhage, n (%)	2	(8.3)
Post operative complication, n (%)	1	(4.2)
Other, n (%)	1	(4.2)
Unknown, n (%)	6	(25.0)
Median survival from the time of rupture - in months, 95% CI in months		
Overall (n = 32)	1.4	(0-6.0)
Surgical intervention (n = 12)	15.5	(8.7-22.2)
Non-resectional type (n = 5)	15.5	(9.7-21.3)
Resectional type (n = 7)	10.8	(*)
Supportive care (n = 20)	0.4	(0.2-0.5)

*Survival estimates cannot be computed since one case is still alive and three cases have been lost to follow-up.

out in all except one whose rupture necessitated an emergency laparotomy with an extended left hepatic lobectomy. Another two cases also received only supportive treatment and, after clinical condition was stabilized, received chemotherapy as palliation for HCC. The remaining cases received exclusively supportive care for HCC rupture and HCC itself.

Staging classification and treatment for each group are summarized in Table 1. Causes of death and patient status for the ruptured group are shown in Table 2. Tumor rupture was the major cause of death in our patients, but it did not cause death in any surgically treated patients. All patients experienced one episode of tumor rupture, even among those expiring from this event.

Active bleeding was revealed in 9 cases during exploratory laparotomies. Among these, tumor resection was performed in 4, packing and suturing of bleeding sites in 3, and hepatic artery ligation in 2. Hemorrhage was successfully con-

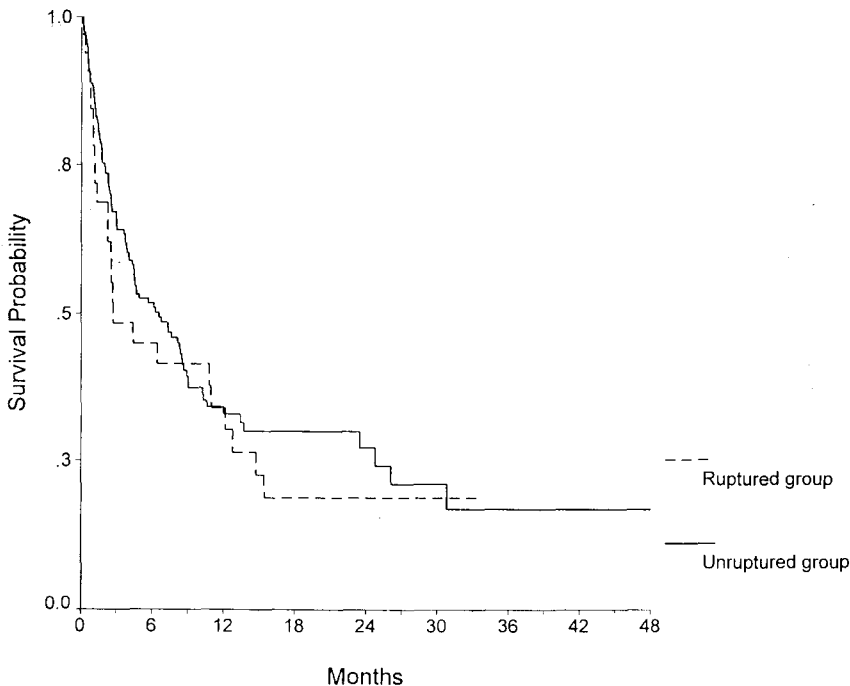


Fig. 1. Survival probability of the ruptured (n = 32) and unruptured (n = 274) groups. Median 2.7 and 6.6 months respectively.

trolled in all and rebleeding did not occur. There were seventeen hospital deaths (53.1%) most of which (14/17, 82.3%) occurred in the patients treated supportively. Nine deaths resulted from the rupture. Two patients who had undergone major hepatic resections died 10 and 24 days after operations as a result of liver failure and respiratory failure resulting from bilateral pulmonary metastases, respectively. One patient treated with suturing and packing died of liver failure 22 days later.

Overall median survival of patients with tumor rupture was 2.7 months (95% CI, 0-5.9 months) from the time of diagnosis. It was not significantly different from that of patients without rupture (median = 6.6 months; 95% CI, 4.0-9.1 months) ($P = 0.4605$) (Fig. 1). Median survival time from the time of rupture was 1.4 months (95% CI, 0-6.0 months) (Fig. 2).

Among the ruptured group, we classified the patients according to treatment received consisting of surgery (resectional and non-resectional types) and supportive care. The patients treated

with surgical intervention survived longer than those receiving supportive care alone (median = 15.5 months; 95% CI, 8.7-22.2 months and median = 0.4 months; 95% CI, 0.2-0.5 months, $P = 0.0027$) (Fig. 3). In addition, we could demonstrate survival difference between the resectional subgroup and the supportive group (median = 10.8 months and median = 0.4 months; 95% CI, 0.2-0.5 months, $P = 0.0300$) and between the non-resectional surgical subgroup and the supportive group (median = 15.5 months; 95% CI, 9.7-21.3 months and median = 0.4 months; 95% CI, 0.2-0.5 months, $P = 0.0209$). No survival difference resulting from the resectional and non-resectional surgical treatments could be demonstrated ($P = 0.9274$) (Fig. 4).

Three patients have survived for more than 30 months after surgical treatments - two emergency non-resectional surgeries and one emergency left lateral segmentectomy. The first two patients then had chemotherapy with and without delayed extended left hepatic lobectomy for HCC treatment.

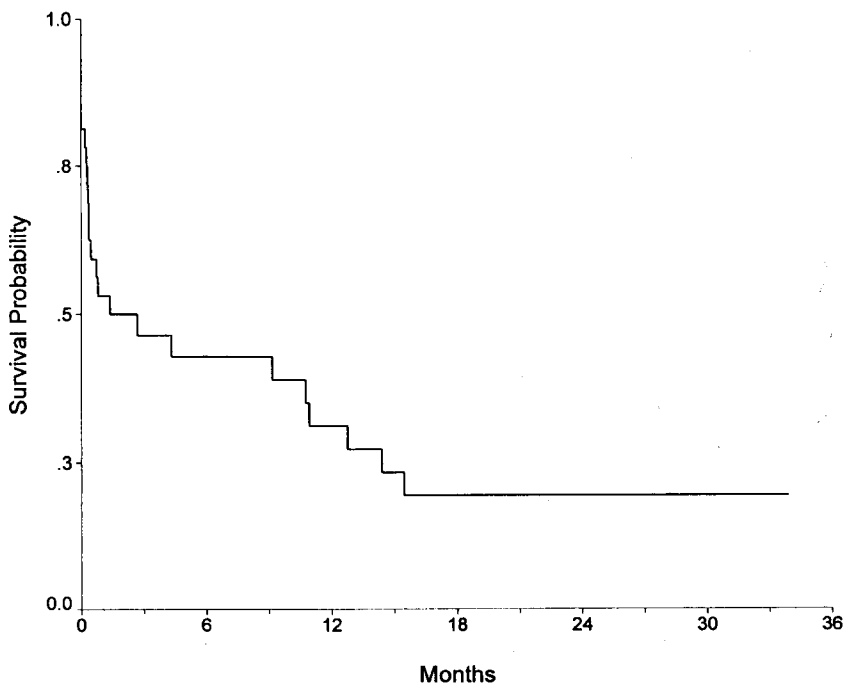


Fig. 2. Probability of overall survival from the time of tumor rupture. Median 1.4 months.

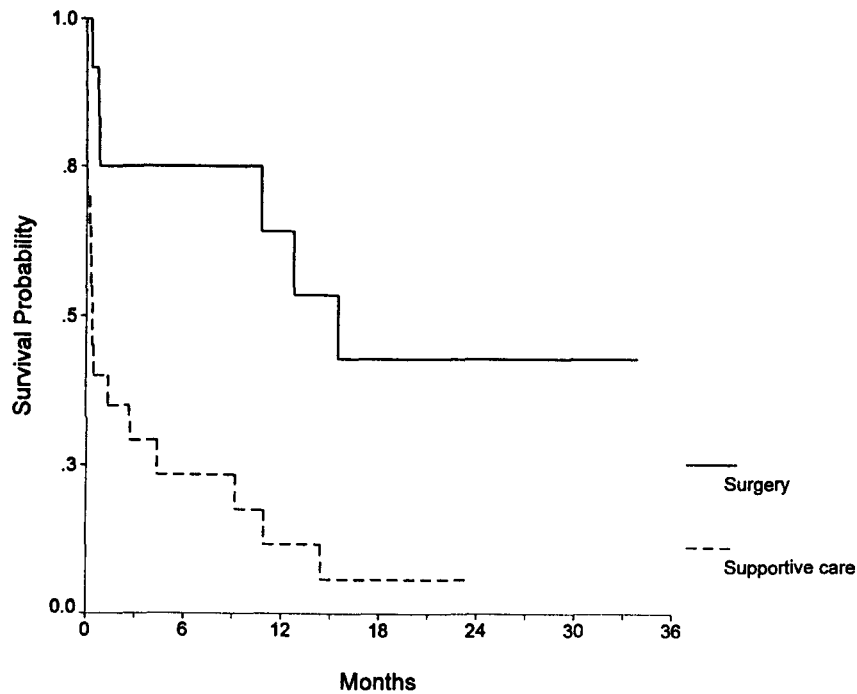


Fig. 3. Probability of survival by treatment received. Difference between the two groups was statistically significant at $P = 0.0027$.

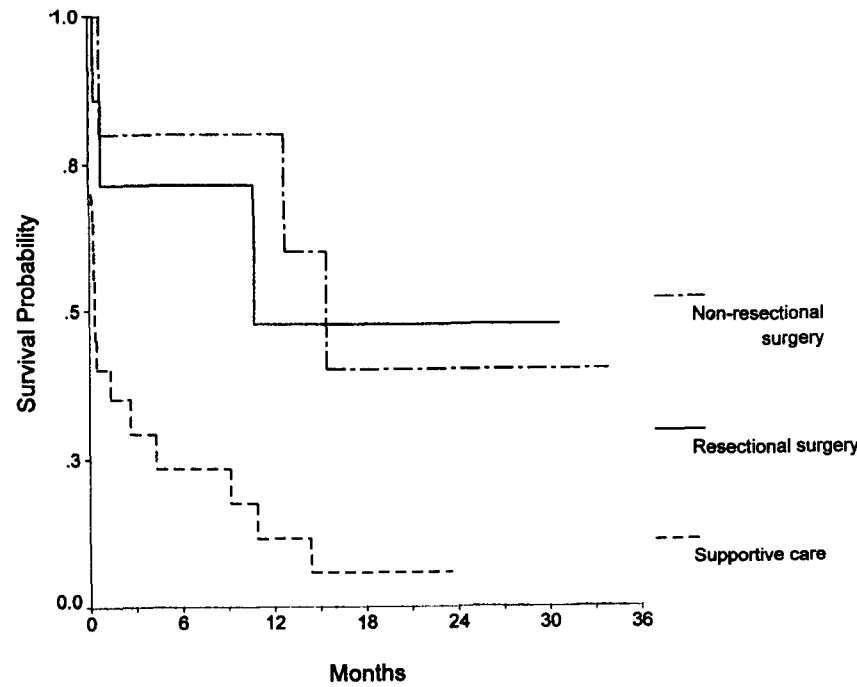


Fig. 4. Survival probability of the resected and non-resectional surgical subgroups. Median 10.8 and 15.5 months respectively.

DISCUSSION

Tumor rupture accompanied by hemorrhagic shock is implicated in facilitating liver failure which is the major cause of death of patients with HCC. It causes hypoxia of the liver which leads to disturbance of the electron transport system and subsequent loss of NADH redox reactions⁽¹⁰⁾. There have been attempts to establish a standard care treatment for ruptured hepatocellular carcinoma^(3,4,6,11-43). Because of various factors involved including tumor extent, liver function reserve, patency of portal vein, and performance status, each patient must be scrutinized for appropriate method of care. However, surgery or tumor resection, if applicable, has been stated to be the treatment of choice^(3,11-25) particularly when a second-stage hepatectomy could be performed^(13, 15,19,21,23). Long-term survival with up to an 8 - year disease free period after hepatic resection for HCC rupture has recently been reported^(24,25).

There were no differences in the age incidence and sex between HCC with and without rupture. This was in contrast with a previous Thai series which revealed male preponderance in HCCs with rupture⁽⁴⁾. Associated cirrhosis was more common in ruptured HCCs ($P = 0.01988$). This agreed with other series, one of which also proposed the hypothesis that obstruction of hepatic vein tributaries draining the tumor-bearing area and portal hypertension due to pre-existing cirrhosis and HCC played important roles in the pathogenesis of spontaneous HCC rupture^(4,22). The severity of portal hypertension is even increased in cirrhosis with HCC as a result of direct hepatoportal arterio-venous shunt in HCC^(44,45).

Our study revealed no significant survival difference between patients with and without tumor rupture despite one third (34.4%) of the patients with HCC rupture benefited from the tumor rupture which emerged as the first clinical presentation of HCC. These patients were diagnosed with HCC at an early stage of disease and could receive appropriate therapy, especially hepatic resection (50% resectability at that time).

Tumor rupture was the major cause of death of these patients whereas liver failure and upper GI hemorrhage were the main causes of death in the unruptured group in our series as well as in ruptured group in other series⁽¹⁹⁾.

Effective hemostasis was achieved in all cases managed surgically. One Chinese series reported a bleeding control rate of 92.3 per cent in tumor resected patients⁽²⁾. For conventional procedures (suturing and packing), successful rates of 62.5 per cent and 20 per cent were also reported from this Chinese and previous Thai series respectively compared with higher rates of 100 per cent and 92.3 per cent for hepatic artery ligation^(2,4).

One patient undergoing emergency transcatheter arterial embolization survived for only 14 days which was much shorter than those from other series (average survival time 3 -7 months)^(19, 26-29). The supportive group had a poor prognosis with median survival time of 0.4 months which was not different from other series (average survival time was 0.4 -1.5 months)^(4,19,27).

The patients who underwent surgical intervention survived longer than those who received supportive care alone. After having had surgical treatments, the majority of our patients survived the following 8.7-22.2 months which was similar to that from previous French and Japanese studies^(6,19). One patient who had undergone a second-stage right hepatic lobectomy survived the following 28 months before becoming lost to follow-up. Survival time after a second-stage hepatectomy of over 60.8 months has also been reported⁽¹⁹⁾. The surgically managed group had its acute bleeding treated and the tumor-resected subgroup even received specific treatment for the tumor as well. Survival benefit over the supportive group could still be demonstrated from both surgical subgroups. However, selection bias among the surgically managed group with favorable prognosis also partly affected the treatment outcome.

In conclusion, we recommend that surgical intervention, if applicable, should be performed in managing rupture of HCC.

REFERENCES

- Alpert ME, Hutt MSR, Davidson CS. Primary hepatoma in Uganda. *AM J Med* 1969; 46: 794-802.
- Ong GB, Taw JL. Spontaneous rupture of hepatocellular carcinoma. *Br Med J* 1972; 4: 146-9.
- Nagasue N, Inokuchi K. Spontaneous and traumatic rupture of hepatoma. *Br J Surg* 1979; 66: 248-50.
- Chearanai O, Plegvanit U, Asavanich C, Damrong-sak D, Sindhvanada K, Boonyapisit S. Spontaneous rupture of primary hepatoma. *Cancer* 1983; 51: 1532-6.
- Cavallari A, Vivarelli M, Bellusci R, De Raffaele E, Nardo B, Gozzetti G. The intraperitoneal bleeding of hepatocellular carcinoma. *Chir Ital* 1994; 46: 45-9.
- Le Neel JC, De Cervens T, Comy M, Dupas B, Letessier E, Mirallie E. Ruptured hepatocellular carcinoma: Report of 20 cases and review of the literature. *Chirurgie* 1994; 120: 380-4.
- Fan ST, Wong J. Hepatocellular carcinoma in the East. In: Terblanche J. *Hepatobiliary malignancy: Its multidisciplinary management*. Avon: The Bath Press, Inc., 1994: 169-88.
- Beahrs OH, Earl Henson D, Hutter RVP, Kennedy BJ. Liver (including intrahepatic bile duct). In: Beahrs OH, Earl Henson D, Hutter RVP, Kennedy BJ. *Handbook for staging of cancer from the manual for staging of cancer, American Joint Committee on cancer and TNM Committee of the International Union Against Cancer*. 4th ed. Philadelphia: J.B.Lippincott Company, 1993: 107-10.
- Okuda K, Ohtsuki T, Obata H, et al. Natural history of hepatocellular carcinoma and prognosis in relation to treatment: Study of 850 patients. *Cancer* 1985; 5: 918-28.
- Ozawa K, Nakatani H, Shimahara Y. Shock and metabolism. *Proceedings of a Seminar on Anesthesia and Reanimation* 1985; 16: 35-54.
- Kawara A, Yamashita C, Yasuda T. A case of hepatic artery ligation for hepatocarcinoma rupture: Its biochemical and histological changes. *Shindan to Chiryō [Igaku Symposium]* 1979; 67: 191-4.
- Tabuse K, Katsumi M, Kobayashi Y. Indication of microwave coagulation therapy against spontaneous primary hepatocarcinoma ruptures and the significance. *Nippon Shokaki Geka Gakkai Zasshi* 1982; 15: 1196-202.
- Takada N, Sano H, Ohmura T. Spontaneous ruptures of primary malignant tumor of the liver. *Hokkaido Geka Gakkai Zasshi* 1983; 28: 69-73.
- Miyazaki K, Nishi J, Uetsuji M. A study of ruptured primary hepatocarcinoma cases with massive intraperitoneal hemorrhage. *J Tokyo Women's Med Coll* 1983; 53: 801-4.
- Yoshikawa T, Yoshida H, Hotta A. Treatment of acute abdominal type hepatocarcinoma. *Nippon Shokaki Geka Zasshi* 1983; 16: 53-7.
- Kawaguchi S, Nagata K, Ikeda K. Internal and surgical treatment of spontaneous rupture of hepatocarcinoma. *Kyukyū Igaku* 1984; 8: 215-20.
- Sugawara K, Wada T, Imura Y. Hepatic rupture in cancer of the liver. *Rinsho Geka* 1985; 40: 357-62.
- Lai EC, Wu KM, Choi TK, Fan ST, Wong J. Spontaneous ruptured hepatocellular carcinoma: An appraisal of surgical treatment. *Ann Surg* 1989; 210: 24-8.
- Miyamoto M, Sudo T, Kyama T. Spontaneous rupture of hepatocellular carcinoma: A review of 172 Japanese cases. *Am J Gastroenterol* 1991; 86: 67-71.
- Alonso M, Reyes G, Galera MJ, Allende L. Hemoperitoneum caused by spontaneous rupture of hepatocarcinoma: Apropos of 8 cases. *J Chir (Paris)* 1991; 128: 130-2.
- Inoue S, Nagao T, Wakabayashi T, et al. Spontaneous rupture of hepatocellular carcinoma: An approach with delayed hepatectomy. *Surg Today* 1992; 22: 474-80.
- Cherqui D, Panis Y, Rotman N, Fagniez PL. Emergency live resection for spontaneous rupture of hepatocellular carcinoma complicating cirrhosis. *Br J Surg* 1993; 80: 747-9.
- Xu HS, Yan JB. Conservative management of spontaneous ruptured hepatocellular carcinoma. *Am Surg* 1994; 60: 629-33.
- Chen MF, Hwang TL, Jeng LB, Jan YY, Wang CS. Clinical experience with hepatic resection for ruptured hepatocellular carcinoma. *Hepatogastroenterology* 1995; 42: 166-8.
- Shirabe K, Kitamura M, Tsutsui S, Maeda T, Matsumata T, Sugimachi K. A long-term survivor of ruptured hepatocellular carcinoma after hepatic resection. *J Gastroenterol Hepatol* 1995; 10: 351-4.
- Sato Y, Fujiwara K, Furui S, et al. Benefit of transcatheter arterial embolization for ruptured hepatocellular carcinoma complicating liver cirrhosis. *Gastroenterology* 1985; 89: 157-9.
- Hirai K, Kawazoe Y, Yamashita K. Transcatheter arterial embolization for spontaneous rupture of hepatocellular carcinoma. *Am J Gastroenterol* 1986; 81: 275-9.
- Soyer P, Van Beers B, Gofette P, Zeitoun G, Pringot J, Levesque M. The role of embolization and chemo-embolization in the emergency treatment of hemoperitoneum caused by spontaneous rupture of hepatocellular carcinoma. *Gastroenterol Clin Biol* 1993; 17: 643-8.
- Boyer JC, Zins M, Vilgrain V, et al. Hemoperitoneum caused by spontaneous rupture of hepatocellular carcinoma: Value and prognostic factors of hepatic artery embolization. *J Radiol* 1995; 76:

30. Kou TP, Yang ZT, Lin WL. Twelve cases of rupture of hepatoma. *Taiwan Hsueh Hui Tas Chih* 1970; 69: 425-36.
 31. Kokka R, Seppala A, Huttunen R, Kairaluoma M, Sutnen S, Larmi T. Spontaneous rupture liver tumours. *Br J Surg* 1976; 63: 715-7.
 32. The Liver Cancer Study Group of Japan. Survey and follow-study of primary liver cancer in Japan, report 7, 1983: 22.
 33. Nouchi T, Nishimura M, Maeda M, Funatsu T, Hasumura Y, Takeuchi J. Transcatheter arterial embolization of ruptured hepatocellular carcinoma associated with cirrhosis. *Dig Dis Sci* 1984; 29: 1137-41.
 34. Naito A, Ono K, Tarumi T. A case of hepatocarcinoma rupture with favorable prognosis using combined therapy of intermittent hepatic arterial blockage and anticancer agent. *Shokaki Geka* 1984; 7: 1677-81.
 35. Oyake E, Kariya M, Chayama K. Emergency TAE therapy for hepatocarcinoma rupture. *Kanzo* 1986; 27: 1094-104.
 36. Takekawa S, Suzuki K, Takahashi G. Diagnosis of spontaneous rupture of hepatocarcinoma and arterial embolization therapy. *Kyukyu Igaku* 1986; 9: 687-99.
 37. Miyazaki M, Udagawa I, Koshikawa H, et al. Influence of hepatic ischemia on liver regeneration following hepatectomy, with special reference to the therapeutic choice for ruptured hepatoma. *Gan No Rinsho* 1989; 35: 787-92.
 38. Mori T, Masuda T, Shimano K, et al. Spontaneous rupture of the metastatic nodule on the peritoneal surface secondary to hepatocellular carcinoma. [letter] *J Clin Gastroenterol* 1991; 13: 594-6.
 39. Soyer P, Levesque M, Zeittoun G, Hassen CS. Hemoperitoneum caused by spontaneous rupture of hepatocellular carcinoma: Role of hepatic artery embolization in the therapeutic procedure. *J Radiol* 1991; 72: 287-90.
 40. Sunderland GT, Chisholm EM, Lau WY, Chung SC, Leung WT, Li AK. Alcohol injection: A treatment for ruptured hepatocellular carcinoma. *Surg Oncol* 1992; 1: 61-3.
 41. Corr P, Chan M, Lau WY, Metreweli C. The role of hepatic artery embolization in the management of ruptured hepatocellular carcinoma. *Clin Radiology* 1993; 48: 163-5.
 42. Abramsohn R, Witz E, Halevy A, Manor H. Spontaneous rupture of a hepatocellular carcinoma successfully managed by embolization of the hepatic artery. *Harefuah* 1993; 124: 68-71.
 43. Goel AK, Sinha S, Kumar A, Chattopadhyay TK. Spontaneous hemoperitoneum due to rupture of hepatocellular carcinoma. *Trop Gastroenterol* 1993; 14: 152-5.
 44. Dork W. Role of increased hepatic arterial blood flow in the portal hypertension of cirrhosis. *Trans Assoc Am Physicians* 1942; 57: 302.
 45. Nagasue N, Inokuchi K, Kobayashi M, Sakee M. Hepatoportal arteriovenous fistula in primary carcinoma of the liver. *Surg Gynecol Obstet* 1977; 145: 504-8.
-

ภาวะแทรกของโรคมะเร็งตับชนิดเฮปาทอเซลลูลาร์ คาร์ซิโนมา ในโรงพยาบาล จุฬาลงกรณ์: การศึกษาย้อนหลังในผู้ป่วย 32 ราย

อรรถพล ปวโรตม์, พ.บ.*, นรินทร์ วรวิทย์, พ.บ.*

ภาวะแทรกของโรคมะเร็งตับชนิด hepatocellular carcinoma พบได้บ่อยในกลุ่มประเทศที่มีความชุกของโรคสูง รวมทั้งประเทศไทย ได้มีความพยายามในการค้นคว้าวิธีการรักษาภาวะนี้ให้ได้ผลดีที่สุด คณะผู้ทำการศึกษาศึกษาถึงผลของการรักษาด้วยวิธีการต่าง ๆ ในผู้ป่วยที่มีภาวะดังกล่าว โดยศึกษาย้อนหลังจากประวัติผู้ป่วย ตั้งแต่เดือนมกราคม พ.ศ. 2532 ถึง เดือนมิถุนายน พ.ศ. 2540 พบว่า ร้อยละ 10.4 (32/306) ของผู้ป่วยโรคมะเร็งตับชนิด hepatocellular carcinoma เกิดภาวะแทรกของก้อนมะเร็งตลอดระยะการดำเนินของโรค อัตรารอดชีวิตเฉลี่ยโดยรวมของผู้ป่วยที่เกิดภาวะนี้ คือ 2.7 เดือน (ช่วงเชื่อมั่น 95% 0-5.9 เดือน) หลังจากได้รับการวินิจฉัย ซึ่งไม่แตกต่างจากอัตราอดชีวิตเฉลี่ย ของกลุ่มผู้ป่วยที่ไม่เกิดภาวะดังกล่าว (อัตราอดชีวิตเฉลี่ย 6.6 เดือน, ช่วงเชื่อมั่น 95% 4.0-9.1 เดือน) ($P = 0.4605$) ในกลุ่มผู้ป่วยที่มีภาวะแทรกของก้อนมะเร็งนั้น กลุ่มผู้ป่วยที่ได้รับการผ่าตัด รอดชีวิตนานกว่ากลุ่มผู้ป่วยที่ได้รับการรักษาแบบประคับประคอง (อัตราอดชีวิตเฉลี่ย 15.5 เดือน; ช่วงเชื่อมั่น 95%, 8.7- 22.2 เดือน และ อัตราอดชีวิตเฉลี่ย 0.4 เดือน; ช่วงเชื่อมั่น 95%, 0.2-0.5 เดือน ตามลำดับ, $P = 0.0027$) กลุ่มผู้ป่วยย่อยที่ได้รับการผ่าตัดก้อนมะเร็งออก และ กลุ่มผู้ป่วยย่อยที่ได้รับการผ่าตัดเพื่อแก้ไขเฉพาะภาวะตกเลือดในช่องท้อง รอดชีวิตนานกว่ากลุ่มผู้ป่วยที่ได้รับการรักษาแบบประคับประคอง ($P = 0.0300$ และ $P = 0.0209$ ตามลำดับ) โดยสรุป ภาวะแทรกของโรคมะเร็งตับชนิด hepatocellular carcinoma ควรได้รับการรักษาโดยวิธีการผ่าตัด หากผู้ป่วยอยู่ในสภาพที่สามารถรับการผ่าตัดได้

* หน่วย Medical Oncology, ภาควิชาอายุรศาสตร์, คณะแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย, กรุงเทพฯ 10330