

Intraperitoneal Seeding from Hepatoma

P. N. Kim, I. Y. Kim, K. S. Lee

Department of Radiology, Soonchunhyang University Hospital, No. 23-20, Bongmyung-Dong, Chunan, Choongnam, 330-100, Korea

Received: 4 May 1993/Accepted: 21 June 1993

Abstract. We report three cases of intraperitoneal seeding from hepatoma. Manifestation of intraperitoneal seeding from hepatoma were intraperitoneal masses (N = 2) and peritoneal thickening (N = 1). Main vascular feeder to intraperitoneal masses was omental branches of the gastroduodenal artery and/or the superior mesenteric artery.

Key words: Liver, hepatoma—Intraperitoneal seeding.

Hepatocellular carcinoma is the most common primary malignant tumor of the liver. It may invade the portal and hepatic venous system but, although extrahepatic metastasis does occur, intraperitoneal metastases are rare [1]. We report three cases with intraperitoneal seeding of hepatoma.

Case Report

Case 1

A 61-year-old man presented with abdominal discomfort of 10 days duration. Physical examination showed abdominal tenderness and distention. Laboratory findings revealed an elevated alpha-fetoprotein (AFP) (425 ng/ml). Paracentesis revealed bloody ascites with 329.7 ng/ml of AFP and 1.08 ng/ml of carcinoembryonic antigen (CEA). Clinical impression was ruptured hepatoma. Postcontrast computed tomography (CT) revealed low density mass in a cirrhotic liver, nodular peritoneal thickening, infiltrated omentum, and ascites (Fig. 1).

Case 2

A 56-year-old man was admitted with abdominal pain for 1 day. Physical examination showed mild abdominal distention and shifting dullness. Laboratory findings showed elevated AFP level (>300 ng/ml).

Paracentesis yielded bloody ascites. US and CT showed a nodule in the left lobe of the liver and ascites (Fig. 2A and B). Under the impression of ruptured hepatoma, hepatic angiography was done, which demonstrated hypervascular nodules supplied from the left hepatic artery and tumor thrombus in the portal vein (Fig. 2C). After selective catheterization of the left hepatic artery, injection of mixture with 50 mg of Adriamycin (Adria, Dublin, OH, USA), 8 ml of Lipiodol Ultra Fluide (Laboratoire Guerbet, France), and Gelfoam (Upjohn, Kalamazoo, MI, USA) particles was done. Fifty days later the patient was readmitted to our hospital because of rebleeding from hepatoma with abdominal distention. AFP was above 300 ng/ml. Plain chest film showed hematogeneous metastatic nodules in both lower lungs. Hepatic angiograms demonstrated multiple nodules supplied from omental branches of gastroduodenal artery, suggesting peritoneal seeding (Fig. 2D). Hemoperitoneum was not improved in spite of embolization of left hepatic artery and he died on day 25, possibly due to bleeding from intraperitoneal metastatic nodules.

Case 3

A 67-year-old man was referred to our hospital for evaluation of right lower abdominal pain and decreased platelet count (71,000/mm³). Physical examination revealed tenderness and rebound tenderness in the right lower quadrant. The clinical impression was acute appendicitis and surgery was performed. About 400 ml of old blood was found in the pelvic cavity and right paracolic gutter. The appendix was retrocecal and normal in size. A 5 × 4 cm sized mass around the appendix was ruptured without evidence of active bleeding. Small hard masses were found around the appendix, on the anterior peritoneal surfaces and in the omentum. All visible nodules were resected and the microscopic diagnosis was hepatoma. Studies to detect hepatoma were then done. AFP was 93.4 ng/ml and CEA was 2.6 ng/ml. CT revealed an ill-defined low attenuation lesion in segments 5 and 6 of the liver.

Eight months later, he presented with a hard palpable mass in the right abdomen. US and CT showed a lobulated mass with central low density and small satellite nodules in the right abdomen, attached to the previous operative site (Fig. 3A and B). Previous ill-defined nodules in the liver were more enlarged and prominent (Fig. 3C and D). Hepatic and superior mesenteric arteriograms demonstrated multiple hypervascular nodules in the liver and a large mass in right abdomen supplied from omental branches of the gastroduodenal artery and branches of the superior mesenteric artery (Fig. 3E and F). After selection of feeding artery from gastroduodenal artery, injection of mixture with 50 mg of Adriamycin and 25 ml of Lipiodol, followed by 0.5 cc of Contour emboli (Interventional Therapeutics Corporation, San Francisco, CA, USA)

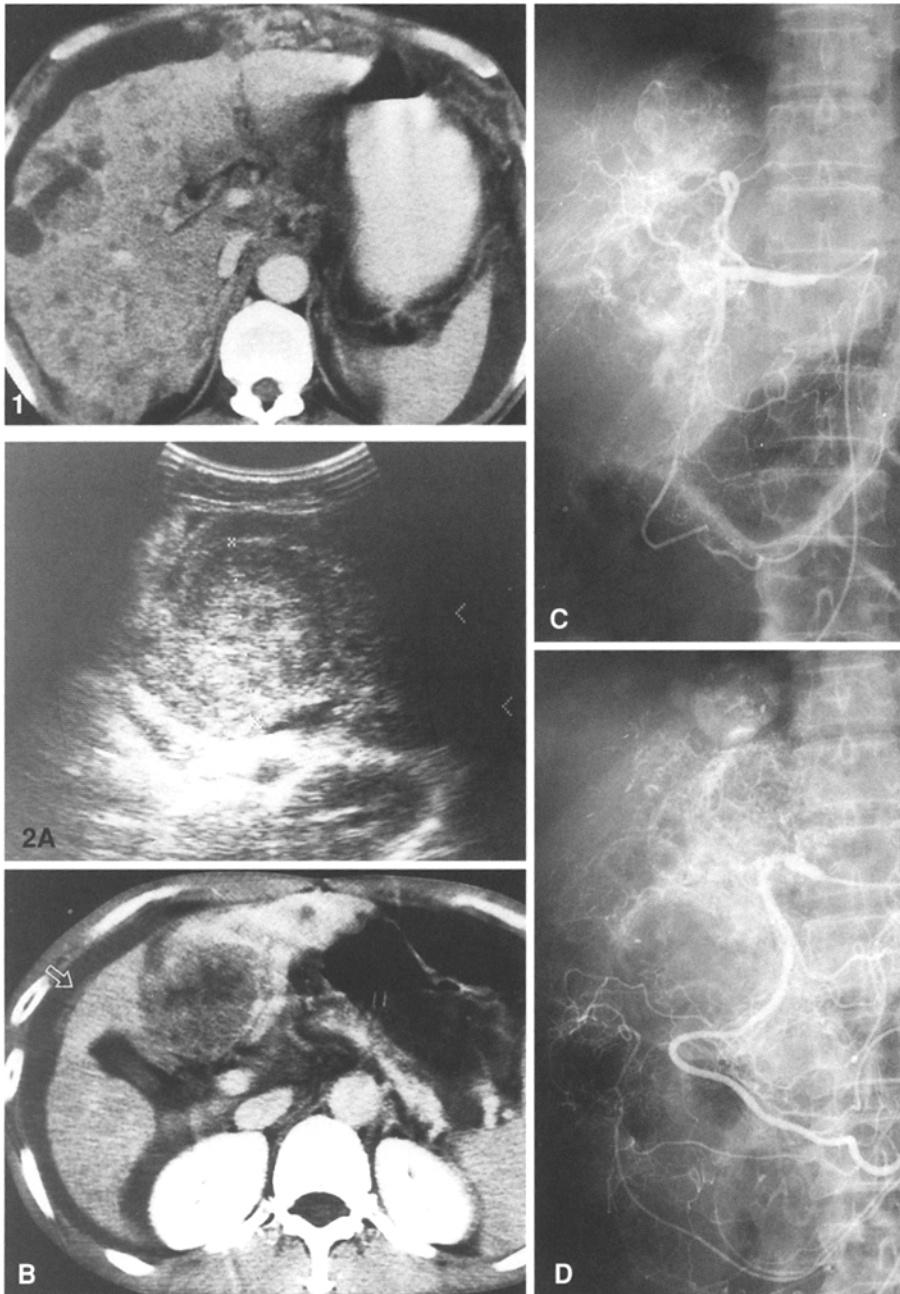


Fig. 1. Case 1. CT demonstrates hepatic nodules, low attenuation in the portal vein suggesting thrombosis, peritoneal thickening, infiltrated omentum, and ascites.

Fig. 2. Case 2. **A** US shows an echogenic mass in the lateral segment of the liver. **B** CT reveals hepatic nodule. High attenuation (*arrow*) in ascites suggests recent hemorrhage. **C** Hepatic angiogram shows hypervascular mass from left hepatic artery and collateral vessels along portal vein represented tumor thrombosis. **D** Follow-up arteriogram after 50 days demonstrates occlusion of hepatic artery and multiple nodules from omental branches of the gastroduodenal artery.

was done. Chemoembolization of the hepatoma was not performed due to decreased hepatic function. One month later, chemoembolization of hepatoma was done using 50 mg of Adriamycin and 10 ml of Lipiodol. On day 7 after chemoembolization, the patient died due to hepatic failure.

Discussion

Bhargava et al. [2] reported the incidence of peritoneal metastasis of hepatocellular carcinoma to be 12%. Extrahepatic metastasis of primary liver cancers occurs

through three routes: hematogenous, lymphogenous, and direct invasion [1]. Therefore, lungs, large veins, and regional lymph nodes were the usual sites involved by hepatoma. Despite the fact that peritoneal implantation is infrequent in hepatoma [1, 3–9], because patients may experience an episode of rupture of hepatoma, implantation of cancer cells should be considered as the mechanism of metastasis. Implantation of cancer is usually manifested by tumor nodules on the serosal surfaces of the bowel and viscera, omentum, and multiple foci in the peritoneal cavity. CT manifestations of

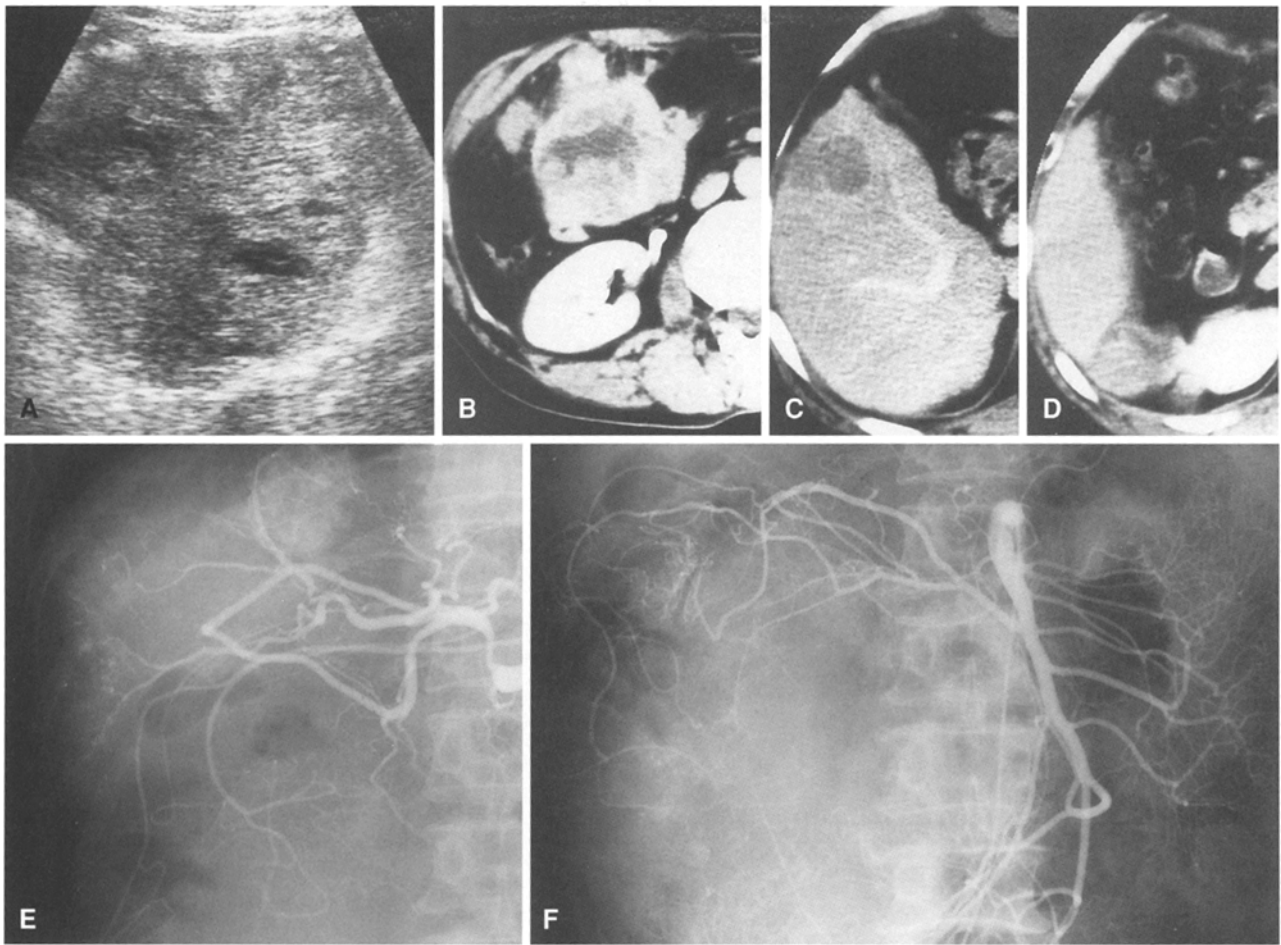


Fig. 3. Case 3. **A** US shows lobulated echogenic mass containing hypoechoic foci in the right abdomen below the liver. **B** CT reveals a well-enhanced mass with central low density and daughter nodules in the right abdomen. **C, D** CT shows low density masses on right lobe of the liver. **E, F** Large intraabdominal mass is supplied from omental branches of gastroduodenal artery and superior mesenteric artery. Also two hypervascular hepatic nodules are seen.

carcinomatosis are ascites, peritoneal thickening with enhancement, and omental changes such as enhancing nodules and omental cake [10]. Small nodular areas with attenuation equal to that of soft tissue on peritoneum or bulky masses along peritoneal surfaces were also noted in carcinomatosis [11]. Two cases in our series showed multiple intraperitoneal nodules and in Case 3, multiple peritoneal and omental nodules along peritoneal surface were noted at operation. In Case 1, ascites, nodular peritoneal thickening, and omental cake were demonstrated. If the hepatic mass had not been detected in Case 1, the diagnosis of carcinomatosis from hepatoma would have been difficult to distinguish from carcinomatosis of other origin. Kubota et al. [12] re-

ported two cases of hepatocellular carcinoma with omental mass after hepatic resection, and in one case the mass was supplied from omental branches of gastroduodenal artery. In our two cases, the main feeding artery of intraperitoneal tumors were omental branches of gastroduodenal artery and/or superior mesenteric artery. Mori et al. [13] reported a case with spontaneous rupture of the metastatic nodule on the peritoneal surface secondary to hepatocellular carcinoma. In our Case 2, in spite of embolization of hepatic artery, hemoperitoneum was not controlled, and suggested bleeding from intraperitoneal nodules.

References

1. Nakashima T, Okuda K, Kojiro M, et al. Pathology of hepatocellular carcinoma in Japan: 232 consecutive cases autopsied in ten years. *Cancer* 1983;51:863-877
2. Bhargava DK, Verma K, Dasarathy S. Laparoscopic and histological features of hepatocellular carcinoma. *Indian J Med Res* 1991;94:424-425
3. Edmonson HA, Steiner PE. Primary carcinoma of the liver. A study of 1-- cases among 48,900 necropsies. *Cancer* 1954;7:462-501

4. Cruickshank AH. The pathology of 111 cases of primary hepatic malignancy collected in the Liverpool region. *J Clin Pathol* 1961;14:120-131
5. Patton RB, Horn RC. Primary liver carcinoma. *Cancer* 1964;17:757-768
6. Ohlsson EGH, Norden JG. Primary carcinoma of the liver: a study of 121 cases. *Acta Path Microbiol Scand* 1965;64:430-440
7. Eduardo LC, Cecilia RS, Jorge AS. Primary carcinoma of the liver in Mexican adults. *Cancer* 1968;22:678-685
8. Anthony PP. Primary carcinoma of the liver: a study of 282 cases in Ugandan Africans. *J Pathol* 1973;110:37-48
9. Chan CH. Primary carcinoma of the liver. *Med Clin North Am* 1975;59:989-994
10. Walkey MM, Friedman AC, Sohotra P, Radecki PD. CT manifestations of peritoneal carcinomatosis. *AJR* 1988;150:1035-1041
11. Hamrick-Turner JE, Chiechi MV, Abbitt PL, Ros PR. Neoplastic and inflammatory processes of the peritoneum, omentum, and mesentery: diagnosis with CT. *Radiographics* 1992;12:1051-1068
12. Kubota S, Inatsuki S, Koito H, Tanada M, Takashima N, Mandai K, Moriwaki S. Two cases of hepatocellular carcinoma with omental mass. *Jpn J Clin Radiol* 1990;35:1077-1080
13. Mori T, Masuda T, Shimono K, Moriyama S, Ikeda T, Umegae S, et al. Spontaneous rupture of the metastatic nodule on the peritoneal surface secondary to hepatocellular carcinoma. *J Clin Gastroenterol* 1991;13:594-596