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A case of necrosis of hepatocellular carcinoma and tumor thrombus in the portal vein induced by transcatheter arterial lipiodol chemoembolization.

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Abstract

A case of hepatocellular carcinoma is reported in which the main tumor, intrahepatic metastases and a tumor thrombus in the portal vein were necrotized completely after Lipiodol chemoembolization. In this case, the tumor thrombus seemed to act as a portal embolus. This phenomenon is interesting because Lipiodol chemoembolization alone usually can not necrotize intra- or extracapsular invasion, intrahepatic metastasis or tumor thrombus in the portal vein. This case is considered to be suggestive of a possible therapy for hepatocellular carcinoma.

KEYWORDS: hepatocellular carcinoma, tumor thrombus, transcatheter arterial embolization

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— Brief Note —

A Case of Necrosis of Hepatocellular Carcinoma and Tumor Thrombus in the Portal Vein Induced by Transcatheter Arterial Lipiodol Chemoembolization

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A case of hepatocellular carcinoma is reported in which the main tumor, intrahepatic metastases and a tumor thrombus in the portal vein were necrotized completely after Lipiodol chemoembolization. In this case, the tumor thrombus seemed to act as a portal embolus. This phenomenon is interesting because Lipiodol chemoembolization alone usually can not necrotize intra- or extra-capsular invasion, intrahepatic metasatasis or tumor thrombus in the portal vein. This case is considered to be suggestive of a possible therapy for hepatocellular carcinoma.

Key words: **hepatocellular carcinoma, tumor thrombus, transcatheter arterial embolization**

Transcatheter arterial embolization (TAE) is used frequently to treat inoperable hepatocellular carcinoma (HCC). It is usually effective aginst main tumors but not so effective aginst extracapsular invasion, intrahepatic metastatic lesions and tumor thrombus in the portal vein. To improve the prognosis after TAE, it is desirable to necrotize the tumor completely. We report herein a case of HCC in which a main tumor, intrahepatic metastases and a tumor thrombus in the portal vein were completely necrotized by Lipiodol chemoembolization. The tumor thrombus in the portal vein seemed to have acted as a portal embolus. This case seemed to be an interesting case which suggests a new direction of therapy for HCC.

A 61-year-old man was admitted to the Okayama University Medical School with a diagnosis of HCC. Computed tomography of the abdomen revealed a low density area of about 5 cm in diameter in the left lateral segment of the liver. Angiography showed that a thin tumor stain in the left lateral segment of the liver and portal vein of the lateral segment was not pictured because of the portal thrombus. After angiography, Lipiodol chemoembolization was performed, in which 20 mg adriamycin suspended in 5 ml Lipiodol was injected from the left hepatic artery. Computed tomography 13 days after Lipiodol chemoembolization revealed the presence of an oval Lipiodol deposition in the left lateral segment and a linear Lipiodol deposition that seemed to lie in a tumor thrombus in the portal vein (Fig. 1). Fifty days after Lipiodol chmoembolization, left lateral segmentectomy of the liver was performed.

Macroscopically, the cut surface showed that the main tumor measuring 5.5 × 3.0 cm was encapsulated and completely necrotic. A tumor

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thrombus extending from the main tumor to the umbilical portion and intra-hepatic metastases were also completely necrotic (Fig. 2). Microscopically, the main tumor, and intra- and extra-capsular invasion was completely necrotic (Fig. 3). A tumor thrombus in the portal vein was also completely necrotic (Fig. 4). The left lateral segment except for the area adjacent to medial

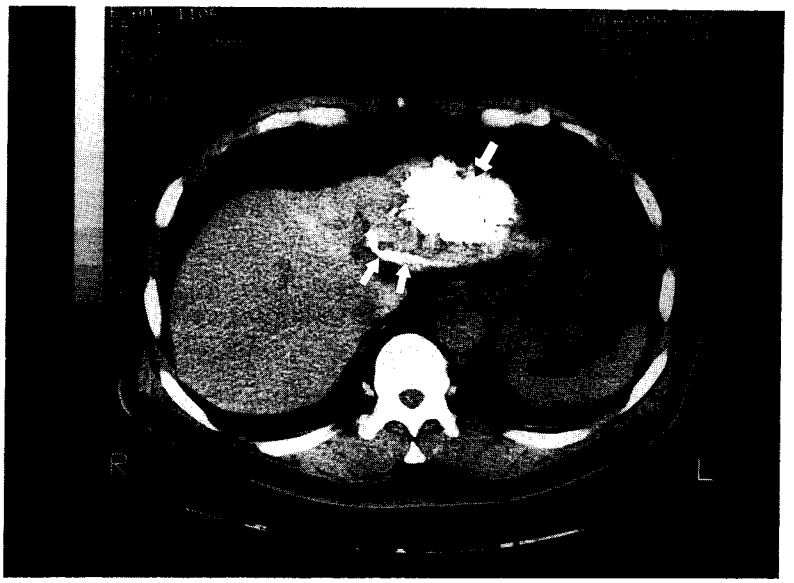

Fig. 1 Lipiodol was detected clearly by Lipiodol CT in the main tumor (⇑) and throughout the tumor thrombus (⇑ ⇑).

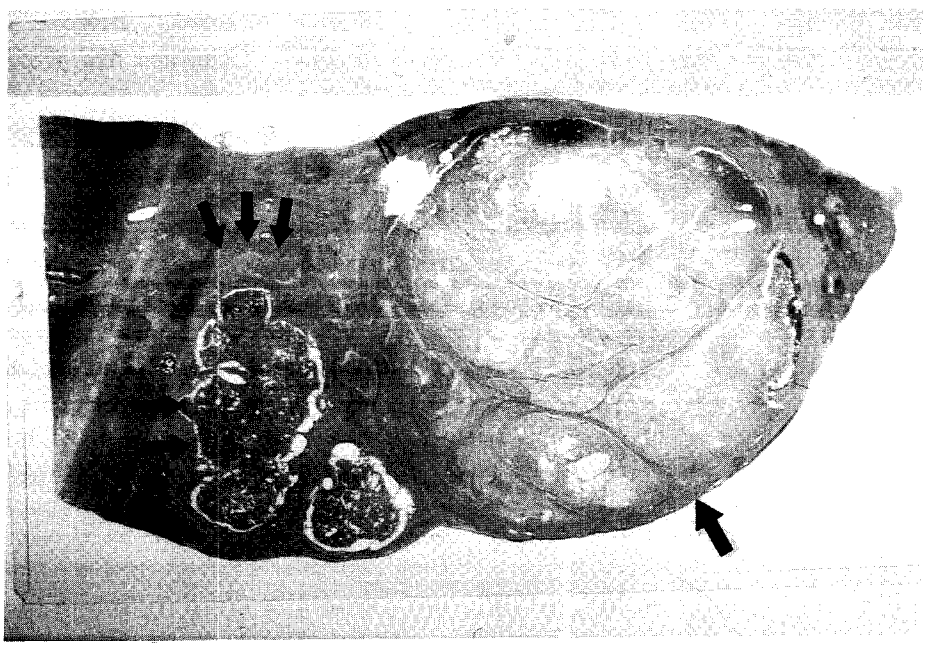

Fig. 2 Macroscopically, the cut surface showed complete necrosis in the main tumor (↑), intrahepatic metastases (↑ ↑) and tumor thrombus in the portal vein (↑ ↑ ↑).

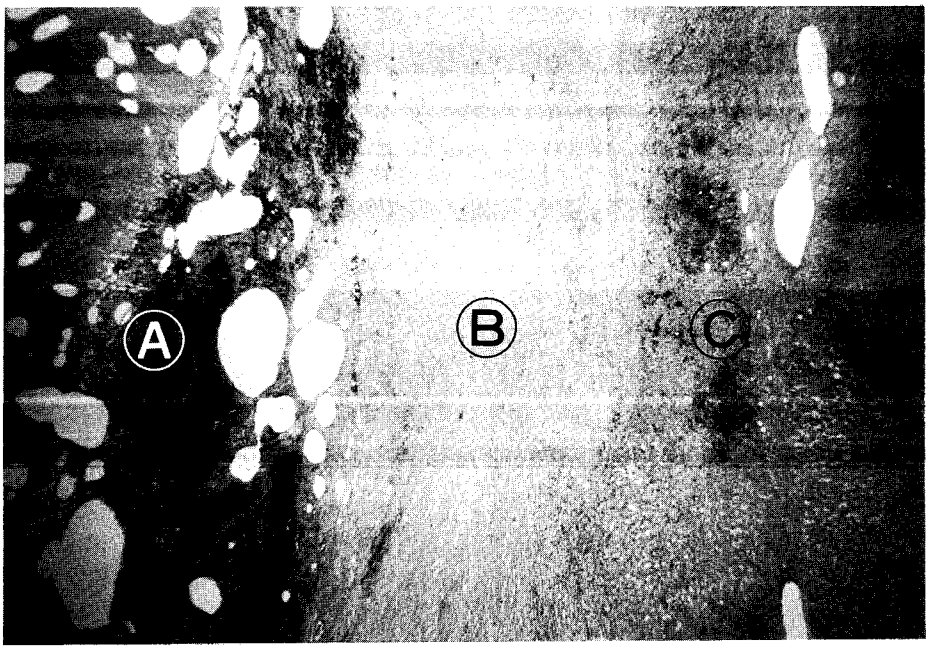

Fig. 3 Complete necrosis of the main tumor (A), and intra-(B) and extra-capsular invasion (C). Degeneration of normal hepatic cells surrounding the tumor was recognized. HE×6.6

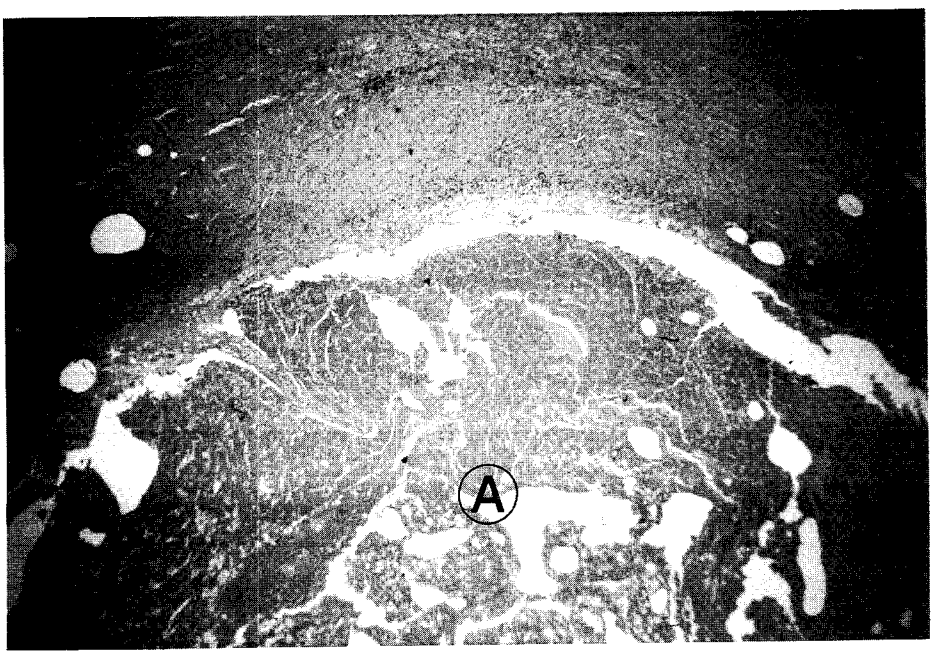

Fig. 4 Tumor thrombus in the portal vein (A) was completely necrosed. HE×6.6

segment has been degenerated. The liver showed chronic inactive hepatitis.

Yamada, *et al.* (1) tried first TAE against HCC in 1977. Since then TAE has become a popular therapy for HCC, especially in inoperable cases. Usually the value of α-fetoprotein decreases remarkably immediately after TAE, but has a tendency to rise again in most cases several months later.

Kojiro (2) examined pathologically specimens resected after TAE and reported that the effects of TAE on the tumor tissue are closely related to the growth patterns of HCC. He divided the growth patterns of HCC into three categories: sinusoidal, replacing and pseudocapsular. In the sinusoidal growth pattern, tumor cells infiltrate the sinusoids at the boundary. This type of HCC does not form a capsule. In the replacing growth pattern, tumor cells proliferate along the liver cell cords, and the sinusoids communicate with the blood spaces of the cancerous tissue. The cancer tissue at the front of the sinusoidal and replacing growth can be nourished by both arterial and portal vein blood. In the pseudocapsular growth pattern, tumor cells grow by compressing non-cancerous tissue, so that a pseudocapsule is formed along the boundary. The effect of TAE differs significantly according to the growth patterns. TAE therapy can not induce complete necrosis at the front of tumor growth in HCC of the sinusoidal and replacing types. The reason is that after TAE such tumor lesions seem to be nourished by portal-vein blood *via* the siousoids, so the tumor tissue left alive continues to grow. TAE therapy can induce complete necrosis in HCC of the pseudocapsular type, because the tumor foci receive only tumoral arterial vessels.

To improve the necrotic effect, Kinoshita, *et al.* (3) developed a combined embolization of hepatic artery and portal vein. TAE was done first, then portal embolization was performed about two weeks later. Liver infarction was avoided by performing portal embolization about two weeks after TAE, by which time recanalization of the embolized arteries usually occurs. Histological examination after combined embolization revealed that the percentage of necrosis was higher than after TAE only, especially in extracapsular invasion, intrahepatic metastases and portal tumor thrombus. However, the authors were unable to obtain complete necrosis of those lesions.

To get complete necrosis, Nakao, *et al.* (4) attempted simultaneous embolization of both the hepatic artery and portal vein. They reported that no viable tumor cells were detected in all five patients examined histologically. Embolization in their study was limited to only the subsegmental region, so that the impact of combined embolization on liver function was nealy the same as that produced when TAE was performed alone.

If more widespread arterial and portal embolization is necessary to get necrosis in the tumor, deterioration of liver function will become a problem. Nevertheless, some cases have been reported, in which combined embolization of the artery and portal vein appeared to be necessary for complete necrosis. Zaitsu, *et al.* (5) reported a case of HCC in which extracapsular invasion, daughter nodules and tumor thrombus in the portal vein were necrosed completely by TAE. Wakasa, *et al.* (6) also reported a similar case of HCC; they used Lipiodol plus TAE instead of the usual TAE. We used Lipiodol chemoembolization without gelatin sponze (Yamanouchi Pharmaceutical Co., Ltd., Japan). In these cases a tumor thrombus embolized the portal vein. Simultaneous embolization of the hepatic artery and portal vein may be a promising therapeutic approach to HCC.

References

1. Yamada R, Sato M and Kawabata M: Hepatic artery embolization in 120 patients with unresectable hepatoma. Radiology (1983) **148**, 397–401.
2. Kojiro M: Histological growth patterns of hepatocellular carcinoma; in Hepatocellular Carcinoma. An Atlas of ICS Pathology, Nakashima and Kojiro eds, Springer-Verlag, Tokyo (1987) pp 81–104.

3. Kinoshita H, Sakai K and Iwasa R: Results of preoperative portal vein embolization for hepatocellular carcinoma. Osaka City Med J (1988) **34**, 115–122.
4. Nakao N, Miura K and Takahashi H: Hepatocellular carcinoma: Combined hepatic, arterial and portal venous embolization. Radiology (1986) **161**, 303–307.
5. Zaitsu H, Taniwaki S and Okuda K: A case of tumor thrombus in the portal vein of hepatocellular carcinoma effects as transcatheter portal branch embolization (TPE). Acta Hepatol Jpn (1988) **28**, 1095–1099.
6. Wakasa K, Sakurai M and Monder M: Necosis of portal tumor embolus of hepatocellular carcinoma by Lipiodol transcatheter chemo-embolization. A case report. Acta Pathol Jpn (1988) **38**, 1363–1367.

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