Case Report

Portal Vein Thrombosis after Radiofrequency Ablation for Recurrent Hepatocellular Carcinoma

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Recurrent hepatocellular carcinoma (HCC) deserves multidisciplinary treatment in addition to surgical resection. Radiofrequency ablation (RFA) is an evolving, localized, thermal ablative treatment for unresectable hepatocellular carcinoma (HCC). Though the preliminary results of RFA in clinical studies are encouraging, its serious complications should not be underestimated. Portal vein thrombosis as a result of direct blood vessel injury by RFA is rarely reported and is potentially fatal in patients with limited liver reserve due to underlying liver cirrhosis. We present a case of portal vein thrombosis as a complication of RFA treatment for recurrent HCC and illustrate its underlying possible mechanism. (Asian J Surg 2003;26(1):50–3)

Introduction

Surgical resection is the standard treatment for hepatocellular carcinoma (HCC), which is one of the most common malignancies in the world. Nonetheless, disease-free survival for patients with curative resection for HCC remains poor due to the high incidence of intrahepatic tumour recurrence (50%-60%)[1,2]. Repeated hepatectomy has been advocated but the resection rate is low (10%-35%)[3,4] due to limited liver reserve and multifocality of the tumours. Among the loco-regional therapies that have been adopted for unresectable HCC, radiofrequency ablation (RFA) is the most recently developed treatment modality. It has proven to be safe, with a low complication rate (0%-12%)[5-7] and is widely practised for unresectable HCC, with a greater than 95% complete tumour ablation rate.[5,8,9] Nevertheless, its serious complications cannot be overlooked, including injury to major intrahepatic blood vessels and bile ducts, liver abscess formation, adjacent organ injury and liver failure. Portal vein thrombosis due to the blood vessel injury by RFA is rarely reported[10] but is potentially fatal in patients with marginal liver reserve. We present a case of portal vein thrombosis as a complication after RFA for recurrent HCC.

Case report

A 43-year-old hepatitis B carrier had an extended right hepatectomy for a 7-cm HCC. He developed intrahepatic tumour recurrence 2 years later, as seen on follow-up computed tomography (CT), and his serum α-fetoprotein concentration increased to 399 ng/dL. The diagnosis was confirmed by fine needle aspiration cytology and there was no evidence of extrahepatic metastasis. The lesion was unresectable because it was close to a main branch of the left portal vein, which was patent, as shown on contrast CT (Figure 1B). There was minimal arterial enhancement within the lesion (Figure 1A), to which transarterial chemoembolization (TACE) was deemed unlikely to be effective due to the scarce blood supply from the hepatic artery. The patient’s liver function remained satisfactory, with an indocyanine green retention test of 11% at 15 minutes.

Open RFA was performed to ablate the tumour. Intraoperatively, there was a 5 x 6-cm tumour mass (Figure 2A) in segment III of the liver, and an adjacent portal vein tumour thrombus (Figure 2B) was identified on intraoperative ultrasonography, which otherwise did not reveal additional tumour within the liver. RFA using the Cool-tip® RF System (Radionics,
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Burlington, MA) was performed under ultrasonographic guidance. No Pringle manoeuvre was applied and complete ablation of the tumour mass together with the portal vein tumour thrombus was achieved with a 1-cm margin. There was no major complication and the patient recovered uneventfully. His liver function returned to the preoperative level 5 days after the procedure and he was discharged on postoperative day 7.

CT was performed 2 weeks after the operation and it confirmed complete tumour ablation. However, there was
thrombosis of the adjacent portal vein branch as shown in the portovenous phase of CT (Figure 3A). The patient’s liver function was deranged, with an increased serum bilirubin concentration (127 mmol/L) and prolonged prothrombin time (17.7 sec). Subsequently, he died of diffuse intrahepatic tumour recurrence 3 months after RFA treatment (Figure 3B).

Discussion

The management of recurrent HCC requires a multidisciplinary approach. Although repeated hepatectomy is the mainstay treatment for recurrent HCC, only a minority (30%-35%) of patients have resectable disease because of limited liver reserve due to previous surgery, underlying liver cirrhosis and multifocality of tumour recurrence.3–11 Treatment of unresectable recurrent HCC relies on various loco-regional therapies for local tumour control. Traditionally, TACE and percutaneous ethanol injection (PEI) have been widely practised with varying degrees of success.12–14 However, TACE may not be feasible for patients with limited liver reserve. Furthermore, its efficacy is restricted by several factors including anatomical changes as a result of previous surgery, collateral blood supply of tumour other than the hepatic artery, damage to non-tumorous liver tissue and accumulated drug toxicity. Likewise, PEI has the disadvantage of a high local recurrence rate (81% after 3-year follow-up),15 due to failure of ethanol to penetrate and dissolve the tumour capsule and intra-tumoral septa. Hence, in recent years, researchers have directed their interest towards another mode of local ablative therapy using thermal energy.

RFA is a newly developed thermal ablative therapy for unresectable HCC. It utilizes high-frequency alternating current (480 kHz) to generate a lethal temperature for the tumour and it is unique in producing a predictable and reproducible thermal ablative lesion. Unlike TACE, RFA does not rely on the arterial blood supply of tumour and it can induce in-situ tumour ablation while preserving maximal normal liver parenchyma. In addition, radiofrequency energy propagates well beyond the tumour capsule to achieve an adequate tumour-negative margin, which is impossible with PEI. Currently, clinical studies involving a total of more than 500 patients treated with RFA have been reported, with a complication rate of 0%-12% and operative mortality rate of 0%-3%.5–9 Curley et al5 reported the largest series of 110 patients who underwent RFA treatment for HCC. There was a 100% complete tumour ablation rate and the ablative site recurrence rate was 3% after a mean follow-up of 19 months. Because of the additional haemostatic effect of RFA, its clinical application can even be extended to HCC with rupture.16

Among the complications of RFA, portal vein thrombosis as a result of direct blood vessel injury is rarely reported. It is potentially fatal in patients with marginal liver reserve. Scudamore et al10 reported a case of portal vein thrombosis after open RFA for a perportal liver tumour using hepatic inflow occlusion (Pringle manoeuvre). This complication reflects the limitation of RFA for liver tumours in close proximity to the portal vein. Because hepatic blood flow carries a significant “heat-sink” effect counteracting the RFA treatment, hepatic inflow occlusion has been shown to enhance the ablative process in animal studies.17,18 However, this manoeuvre may be risky when applied to liver tumours close to the main branch of the portal vein because of the loss of the protective effect of hepatic blood flow on the vein wall against RFA. On the other hand, the completeness of tumour ablation

![Figure 3. Postoperative computed tomographic scan shows complete ablation of the liver tumour and intrahepatic portal vein thrombosis (A) and subsequent diffuse intrahepatic tumour recurrence (B).](image-url)
along the blood vessel is questionable if the Pringle manoeuvre is prohibited in this situation.

Our case illustrates a similar clinical condition to that mentioned. Though we did not apply the Pringle manoeuvre during RFA treatment, the identified tumour thrombus may reduce portal venous blood flow to the extent that the affected portal vein branch became vulnerable to RFA injury. The resulting detrimental effect of portal vein thrombosis was well shown in our patient after RFA treatment. Moreover, the subsequent rapid tumour dissemination in the liver remnant can be explained by the inadequate tumour control by RFA in the presence of tumour invasion into the portal vein. In fact, venous invasion is an independent risk factor for early intrahepatic recurrence of HCC after curative resection in multivariate analysis.19

In conclusion, RFA should be avoided in liver tumours close to the main portal vein branch if there is reduced blood flow due to any cause, such as a portal vein tumour thrombus in our patient. Furthermore, in the presence of a portal vein branch tumour thrombus, RFA may not induce complete tumour control. Thus, it is not an appropriate treatment from the oncological point of view.

References