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CASE REPORT

# The successful treatment of hepatocellular carcinoma arising from congenital hepatic fibrosis using radiofrequency ablation under laparoscopy

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## Abstract

Congenital hepatic fibrosis (CHF), a fibropolycystic disease, is characterized by bile duct malformation, periportal fibrosis, and renal polycystic disease. Although cholangiocellular carcinoma is the primary tumor arising from fibropolycystic diseases, hepatocellular carcinoma (HCC) is extremely rare. In addition, no algorism for determining the optimum HCC treatment has yet been available in cases of fibropolycystic disease due to variations in the background liver and renal conditions. We herein report a patient with HCC arising from CHF that was successfully treated using radiofrequency ablation (RFA) under laparoscopic assistance. A 37-year-old man with CHF was admitted to our hospital for treatment of HCC in 2014. Imaging revealed HCC located in hepatic segments II and VIII with diameters of 28 and 24 mm, respectively. There had been no histories of recurrent cholangitis or renal failure after when CHF was diagnosed in 2003. In addition, esophageal varices were well controlled. We achieved sufficient ablation using a bipolar ablation system without any complications. The post-operative course was uneventful, and the patient was free from HCC for 4 years. Thus, locoregional therapy, including RFA, is acceptable for the treatment of HCC arising from CHF when the background liver and kidney are preserved.

**Keywords** Congenital hepatic fibrosis · Hepatocellular carcinoma · Locoregional therapy · Radiofrequency ablation

## Introduction

Fibropolycystic diseases are congenital disorders and include congenital hepatic fibrosis (CHF), autosomal recessive polycystic kidney disease (ARPKD), bile duct hamartomas, choledochal cysts, and Caroli disease. These disorders affect patients of all ages, from infants to elderly individuals, and present with varying degrees of liver fibrosis, recurrent cholangitis, and renal failure [1].

The fibropolycystic disease CHF was first reported by Kerr in 1961 [2], and 200 cases had been reported by 1988 [3]. CHF is an autosomal recessive disorder and shares a PKHD1 gene mutation with ARPKD [1]. Although CHF and ARPKD are similar clinical entities, CHF tends to be observed in adolescents and young adults involving hepatic manifestations, whereas ARPKD affects infants and involves renal cystic diseases. CHF presents with portal hypertension and/or recurrent cholangitis, with the portal hypertension type being the most common. Histologically, CHF is predominantly characterized by periportal fibrosis and proliferative bile ducts with irregularly shapes.

Liver tumors arising from fibropolycystic diseases are relatively rare. The prevalence of liver tumors in CHF and Caroli disease ranges from 1.0–5.9% to 3.2–7.0%, respectively [4, 5]. Among liver tumors arising from fibropolycystic diseases, cholangiocellular carcinoma is the primary tumor, whereas hepatocellular carcinoma (HCC) is extremely

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rare. Partial hepatectomy and liver transplantation are often selected as a treatment for cholangiocellular carcinoma to remove the tumors as well as potential sources of malignancy. However, the best way to treat HCC has not been determined in patients with fibropolycystic diseases, probably due to the varied background liver and renal conditions.

We herein report our experience of a patient with HCC arising from CHF and the successful treatment of the tumor using radiofrequency ablation (RFA) under laparoscopy. Because information on the course of CHF with HCC is lacking, we reviewed the literature to determine the frequency of HCC and treatment choices for HCC arising from fibropolycystic diseases.

## Case report

A 26-year-old man visited a hospital for further evaluation of low platelet count at his health check-up in April 2003. At that time, he was noted to have esophageal varices, which was consequently treated with endoscopic variceal ligation. He had no family history of liver or kidney diseases, including fibropolycystic diseases. His daily alcohol intake was less than 30 g ethanol per day. Because laboratory tests were negative for viral hepatitis and autoimmune-related hepatitis, liver biopsy was performed in 2003. The histological examination showed the characteristics of CHF, including irregularly dilated bile ducts and severe portal fibrosis. The present case was determined to be portal hypertension type because of the history of esophageal varices and the lack of any experience of cholangitis as well as renal disease.

A liver tumor, detected at hepatic segment II in October 2013, grew in size during the follow-up, so tumor biopsy was performed at the previous hospital in August 2014. The histological examination demonstrated that the tumor was well-differentiated HCC. Then, he was admitted to our hospital for the treatment of HCC in November 2014 when he was 37 years.

On a physical examination, his consciousness was alert. There were no findings of jaundice, ascites, or edema. The liver and spleen were not palpable. Laboratory data showed a slight elevation of hepatobiliary enzymes and a decreased platelet count (Table 1). Hepatitis B surface antigen, hepatitis B core antibody, and hepatitis C virus antibody were negative. Other liver diseases were denied by laboratory tests. Protein induced by vitamin K absence or antagonist II (PIVKA-II) was slightly elevated, but alpha-fetoprotein was within normal. Urine examination did not reveal any abnormalities. Contrast-enhanced abdominal computed tomography (CT) showed that a slight dilatation of the intrahepatic bile ducts, splenomegaly, and collateral veins (Fig. 1a, b). In magnetic resonance imaging (MRI), multiple cystic changes were detected at the peripheral areas of the liver (Fig. 1c) but

**Table 1** Laboratory data on admission

| | | |
|---|---|---|
| Hematology | | |
| WBC | $4.8\times10^3$ | /μl |
| Hb | 15.9 | g/dl |
| Plt | $6.7\times10^4$ | /μl |
| Coagulation test | | |
| PT-INR | 1.15 | |
| PT% | 86 | % |
| APTT | 39.3 | s |
| Blood chemistry | | |
| TP | 7.1 | g/dl |
| Albumin | 4.3 | g/dl |
| T-bil | 1.83 | mg/dl |
| D-bil | 0.2 | mg/dl |
| AST | 42 | U/L |
| ALT | 35 | U/L |
| LDH | 203 | U/L |
| ALP | 231 | U/L |
| γ-GTP | 89 | U/L |
| ChE | 247 | U/L |
| BUN | 12 | mg/dl |
| Cr | 0.85 | mg/dl |
| Na | 142 | mmol/L |
| K | 4.0 | mmol/L |
| Cl | 109 | mmol/L |
| IgG | 959 | mg/dl |
| IgM | 144 | mg/dl |
| ANA | ± | |
| AMA | − | |
| Tumor marker | | |
| AFP | 3 | ng/ml |
| PIVKA-II | 78 | mAU/ml |
| CEA | 2.2 | ng/ml |
| Virus marker | | |
| HBs Ag | (−) | |
| HBc Ab | (−) | |
| HCV Ab | (−) | |
| Urianalysis | | |
| Color | Yellow | |
| pH | 5.5 | |
| Protein | (−) | |
| Glucose | (−) | |
| Uro | (±) | |
| Bilirubin | (−) | |
| Ketone | (±) | |
| O.B | (−) | |

*WBC* white blood cells, *Hb* hemoglobin, *Plt* platelet count, *PT* prothrombin time, *INR* international normalized ratio, *APTT* activated partial thromboplastin time, *TP* total protein, *T-bil* total bilirubin, *D-bil* direct bilirubin, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *LDH* lactate dehydrogenase, *ALP* alkaline phosphatase, *γ-GTP* γ-glutamyl transpeptidase, *ChE* cholinesterase, *BUN* blood urea nitrogen, *Cr* creatinine, *ANA* anti-nuclear antibody, *AMA*

**Table 1** (continued)

anti-mitochondrial antibody, *AFP* alpha-fetoprotein, *PIVKA-II* Protein induced by vitamin K absence or antagonist II, *CEA* carcinoembryonic antigen, *HBsAg* hepatitis B surface antigen, *HBcAb* hepatitis B core antibody, *HCVAb* anti-hepatitis C virus antibody, *Uro* urobilinogen, *O.B* occult blood

not the large bile ducts or the extrahepatic bile duct. Based on laboratory and imaging examinations, we suspected CHF as the primary disorder rather than hamartoma, choledactal cyst, and Caroli disease. In addition, no morphological changes were observed in the kidneys, including cystic lesions.

Regarding the tumors, contrast-enhanced abdominal CT showed tumors at segment VIII in addition to segment II. The size of tumors at hepatic segment II and VIII was in 28 and 24 mm in size, respectively. Both tumors were enhanced during the arterial phase, while the density became low to isodense during the portal venous phase (Fig. 2a–d). MRI revealed the same size of tumors in segments II and VIII. The tumors were enhanced compared to the liver parenchyma during the arterial phase, in a dynamic study using gadolinium-ethoxybenzyl diethylenetriamine pentaacetic acid (Gd-EOB DTPA). In the portal venous phase, the tumors showed a low-intensity lesion with a slightly high-intensity ring. In the hepatobiliary phase, the tumors showed defective areas of Gd-EOB DTPA (Fig. 2e–j). We strongly suspected that the both tumors were HCC from the findings of CT and MRI.

Because the patient rejected to receive partial hepatectomy and the tumor located in segment VIII was difficult to access via the percutaneous approach, we performed laparoscopy for the observation of the liver surface and subsequent RFA under general anesthesia. On laparoscopy, the left lobe was enlarged, while the right one was shrunken. Features of CHF, including collateral vessels, whitish marking, and black–green spots, were observed on the surface of the liver (Fig. 3). We used a multipolar RFA device (Celon POWER System, Olympus, Japan) to ablate the tumors, because both tumors were located close to the liver surface (Fig. 3). Two applicators were inserted at the dorsal portions of the tumor sides, and one was inserted at the ventral portion on the tumor margin under assistance with ultrasonographic guidance (TUS A-300, TOSHIBA, Tokyo, Japan). Under automatic impedance control, the tumors located in segments II and VIII were completely ablated by 19- and 15-min sessions, respectively. On histological examinations, the tumor in segment II was found to be well-to-moderately differentiated HCC (Fig. 4a, b). The tumor in segment VIII was likely well-differentiated HCC (not shown). The background liver showed irregularly dilated bile ducts in the portal area and severe periportal fibrosis (Fig. 4c, d). The post-operative course was uneventful, and he was discharged 5 days after the operation. At present, no recurrence of HCC has been noted.

## Discussion

Malignant hepatic tumors in fibropolycystic diseases are a relatively rare complication. In 2012, Srinath et al. reviewed 1230 patients with fibropolycystic diseases described in

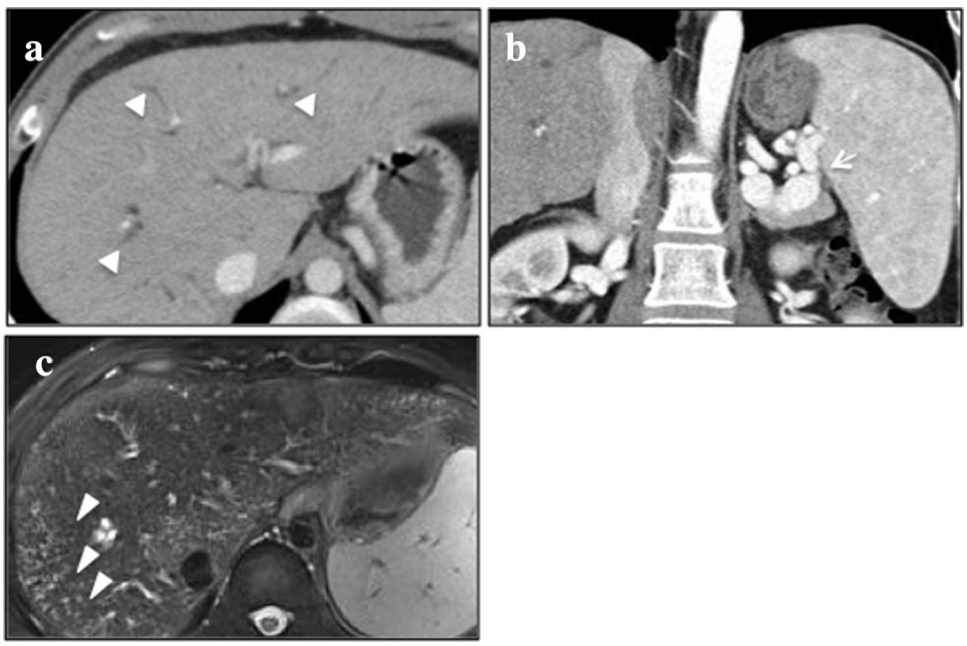
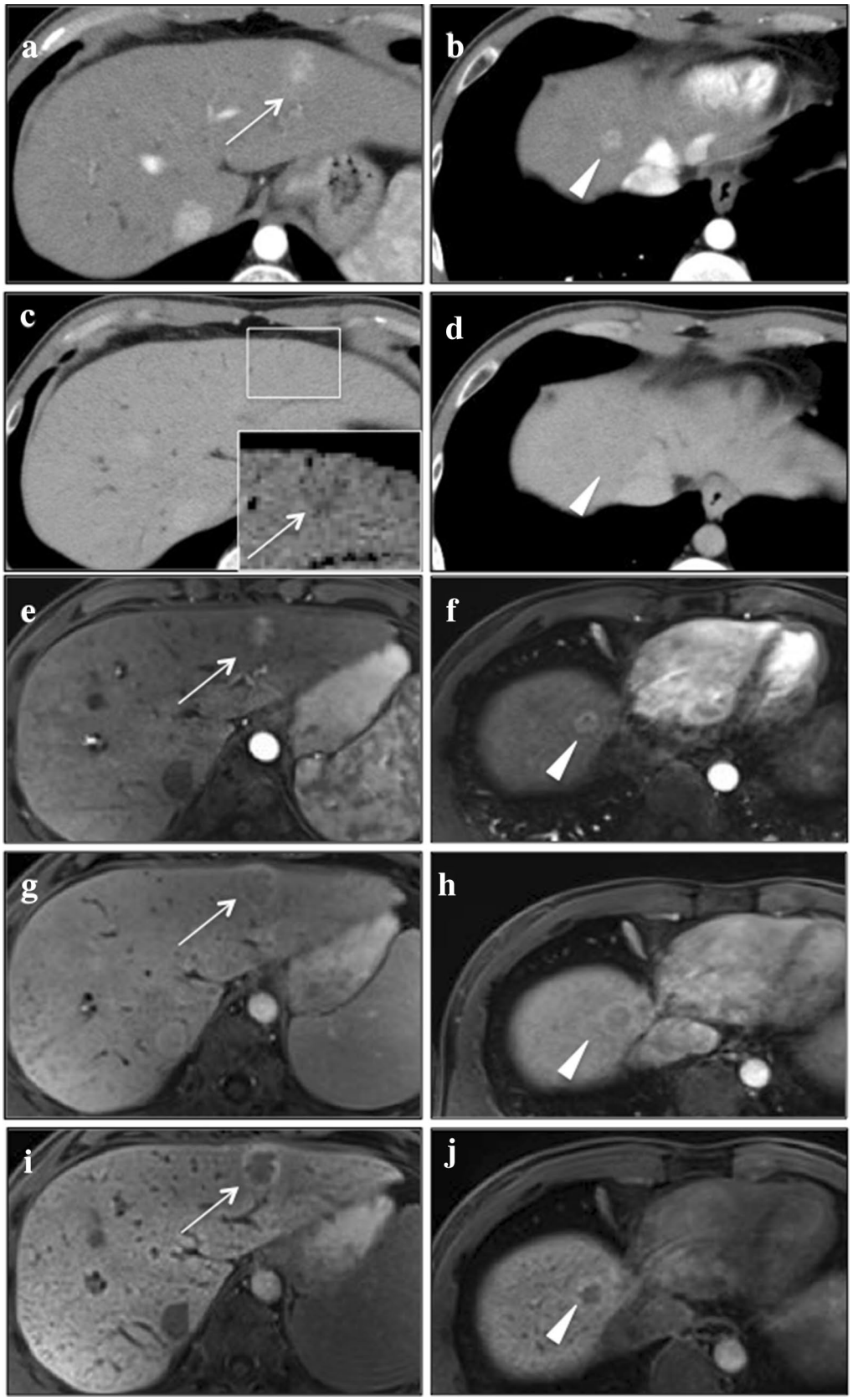



**Fig. 1** Findings of contrast-enhanced abdominal CT and MRI. **a** Intrahepaitc bile ducts are slightly dilatated (arrowheads) on CT scans. **b** Sagittal view shows splenomagaly and dilation of the collateral veins (arrow) on CT scans. **c** On T2-weighted MR imaging, multiple cystic changes are observed as high- intensity areas at the periphery of the right lobe (arrowheads)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◂**Fig. 2** Findings of contrast-enhanced abdominal CT and MRI for tumors. **a–d** Findings of contrast-enhanced abdominal CT. **a, b** Arterial phase images show that both tumors are enhanced by the contrast material (**a** segment II, arrow; **b** segment VIII, arrowhead). **c, d** Portal venous images show that both tumors are low to isodense compared with the surrounding area. Inset in **c** is magnification of the boxed area after making a contrast. The tumor in segment II shows a low-density area (arrow). However, the tumor in segment VIII is isodense even after making a contrast (not shown). **e–j** Contrast-enhanced MRI using Gd-EOB DTPA. The tumors located segment II (**e, g, i**) and VIII (**f, h, j**) are shown arrow and arrowhead, respectively. The both tumors are enhanced compared to the liver parenchyma in the arterial phase (**e, f**). In the portal phase, the tumors show low intensity with a slightly high-intensity ring (**g, h**). In the hepatobiliary phase, the tumors are observed as defective areas (**i, j**)

the literature reported from 1950 to 2010 [5], in which the prevalence of malignant hepatic tumors was 2% (21/1230 patients). Of these 21 patients, 19 had cholangiocellular carcinoma, but HCC were not documented [5]. We, therefore, reviewed the literature (PubMed from 1970 to 2017) to determine the frequency of HCC arising from CHF as well as other fibropolycystic diseases, including hepatic hamartomas and Caroli disease. We collected a total 51 patients with malignant liver tumors after excluding 8 patients with virus hepatitis, alcohol liver injury and hemochromatosis. Among these 51 patients, CHF, hamartomas, Caroli disease, and combination of Caroli disease and CHF were reported in 8, 19, 21, and 3 cases, respectively (Table 2). Of the 8 CHF patients with liver tumors, 5 cases were HCCs (62.5%). In contrast, the rate of HCC arising from hamartomas and Caroli disease was 15.8% (3/19) and 9.5% (2/21), respectively. The reasons for the high prevalence of HCC among patients with CHF are unknown. Liver fibrosis is well known to carry a risk of HCC in case of chronic hepatitis. Indeed, our case had severe fibrosis in the portal areas. Thus, CHF may have a great potential to induce development of HCC than bile duct hamartomas and Caroli disease. In contrast, the majority of malignant tumors arising from hamartomas and Caroli disease were cholangiocellular carcinoma (Table 2). This is because hamartomas and Caroli disease have a tendency to be complicated with recurrence cholangitis, a potential cause of cholangiocellular carcinoma.

At present, five cases of HCCs, including our own patient, arising from CHF have been reported in the literature since 1970 [6–9] (Table 3). We, therefore, determined the characteristics of HCC arising from CHF. The mean age of patients was 43 years, which is younger than that of patients with HCV-related HCC (mean 61 years) [10]. This is because CHF is a congenital disorder frequently affecting patients from childhood. Although fibropolycystic diseases are characterized by recurrent cholangitis, no reports of cholangitis were noted in these five patients with HCC.

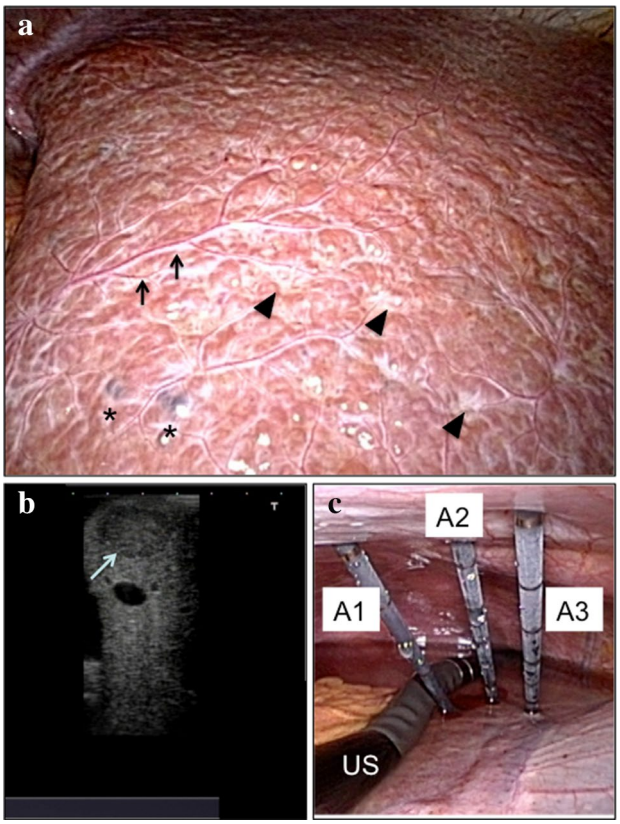


**Fig. 3** Laparoscopic findings and procedures of RFA under laparoscopy. **a** Dilated collateral vessels (arrow), white markings (arrowhead), and "black and green spots" (asterisk) are observed on the surface of the liver. **b** Ultrasonographic imaging of the tumor located in segment VIII shows a low-echoic mass (arrow). **c** Multipolar RFA system is used to ablate the tumors located in the segment VIII. Applicators are inserted at the dorsal portion of the tumor side (A1, A3) and the ventral portion of the tumor edge (A2). US: a probe of ultrasonography

The treatments for HCC developing in CHF patients vary (Table 3). In contrast, partial hepatectomy were selected for the treatment of HCC in patients with other fibropolycystic diseases [11–14]. This is probably due to the benefit of removing the potential source of malignancy caused by recurrent cholangitis. In addition, the sizes of most HCCs exceeded the indication for RFA treatment. Liver transplantation is an alternative treatment for patients with fibropolycystic diseases, because such patients show recurrent cholangitis and portal hypertension-related complications. However, there is a shortage of donors for liver transplantation in Japan. We elected to perform RFA under laparoscopic assistance for the following reasons: (1) the tumor size was suitable for complete ablation; (2) the patient had no history of recurrent cholangitis or renal disease; (3) portal hypertension was well controlled; (4) the percutaneous approach was difficult in

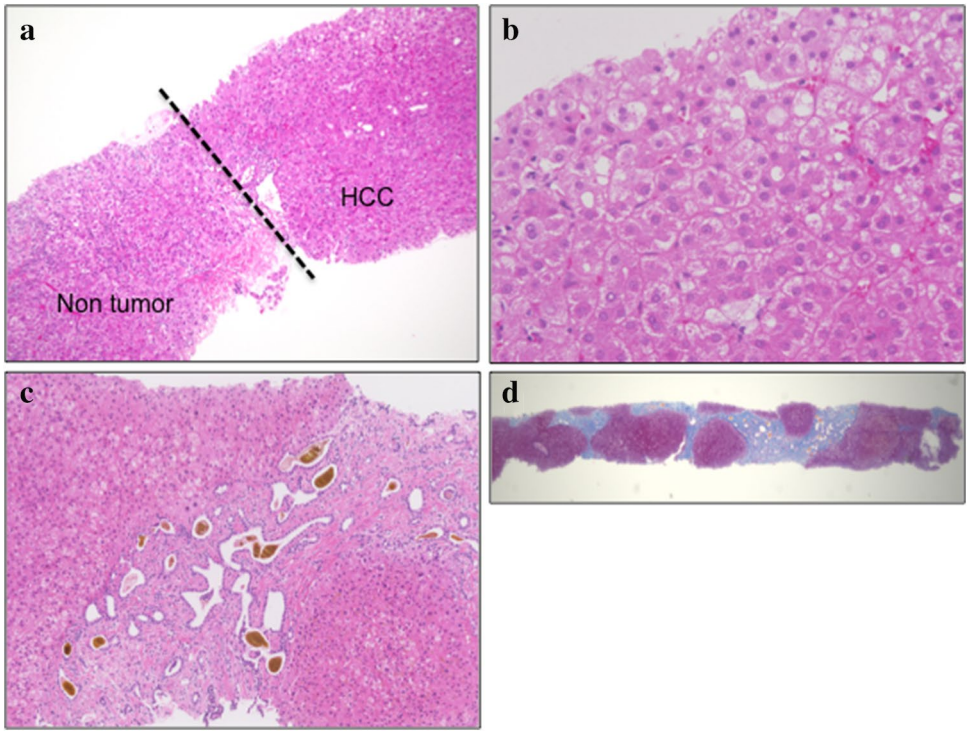


**Fig. 4** Histological findings of the tumor and background liver. **a**–**c** Hematoxylin and eosin staining. **a** Margin of the tumor is shown (×100). **b** Tumor in segment II show high nuclear/cytoplasm ratio and increased cell density. In addition, atypical hepatocytes (tumor cells) are arranged in a trabecular pattern (×400). The tumor is well-to-moderately differentiated HCC. **c** Irregularly dilated bile ducts are observed along with some concentrated bile plugs in the non-tumor areas (×200). There are few inflammatory cells in the parenchyma. **d** Azan staining. Portal areas are enlarged with bridging fibrosis (×20)

**Table 2** Liver tumors arising from fibropolycystic diseases

| | HCC | Cholangiocellular carcinoma | Total |
|---|---|---|---|
| CHF | 5 (62.5%) | 3 (37.5%) | 8 |
| Hamartoma | 3 (15.8%)[a] | 16 (84.2%) | 19 |
| Caroli | 2 (9.5%) | 19 (90.5%) | 21 |
| Caroli + CHF | 0 (0.0%) | 3 (100%) | 3 |
| Total | 10 | 41 | 51 |

[a]Includes one patient with hepatocellular carcinoma (HCC) and cholangiocellular carcinoma

the present case; and (5) the patient rejected hepatectomy. We completed RFA without any complications, including hemorrhaging. In addition, the patient has been free from HCC or other malignancies for more than four years. As we can safely perform bipolar RFA under laparoscopy [15, 16], locoregional treatments, including bipolar RFA, should be considered indicated for HCC arising from CHF, if available. In addition, no-touch bipolar system has been reported to lower intrasubsegmental recurrence of HCC [17]. Thus, bipolar system may have an advantage to prevent the dissemination of tumor if subsequent histological examinations demonstrated that the tumors were non-HCC malignancies, including cholangiocellular carcinoma.

The natural history of CHF with HCC is largely unknown. We were able to follow the present patient for 10 years from the initial diagnosis of CHF until HCC development and then a further 4 years after treatment for HCC. The prognosis of portal hypertension-type CHF is favorable when portal hypertension is well controlled [18]. Indeed, gastrointestinal varices were not observed after the initial treatment for esophageal varices in our patient. However, this prolonged survival of CHF patients may carry a risk of malignant liver tumors, including HCC. We must, therefore, carefully follow up CHF patients with HCC.

In the present case, we were able to confirm the diagnosis of CHF by laparoscopic observation of the liver surface. The findings of the liver surface were compatible to those observed in CHF, including whitish markings, black–green spots, and collateral vessels. Indeed, Komatsu et al. reported that the laparoscopic findings of CHF are characterized by whitish markings, black–green spots, and collateral vessels on the surface of the liver [19]. Whitish markings and black–green spots are associated with portal fibrosis and bile duct dilation, respectively. Black–green spots are suitable sites for a biopsy to make the diagnosis of CHF. In addition, a laparoscopic examination can avoid a risk of bleeding, because collateral veins are well-developed in CHF.

**Table 3** Characteristics of 10 HCCs arising from fibropolycystic diseases

| | First author | Refs. | Age | Gender | Background | PH | Cholangitis | Kidney | Maximum size | Number | Differentiation | Treatment | Prognosis |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Manes | [6] | 69 | M | CHF | Yes | No | Yes | 70 mm | 1 | N/A | Autopsy | |
| 2 | Bauman | [7] | 31 | M | CHF | Yes | No | Yes | 20 mm | Multiple | Well | Transplantation | > 4 years |
| 3 | Ghadir | [8] | 27 | F | CHF | Yes | No | No | 59 × 39 mm | 1 | N/A | RFA | U/K |
| 4 | Kinugasa | [9] | 51 | F | CHF | Yes | No | No | 22 mm | 3 | Well | TACE, RFA, PHx | U/K |
| 5 | Our case | | 37 | M | CHF | Yes | No | No | 28 mm | 2 | Well–moderate | RFA | > 4 years |
| 6 | Heinke | [11] | 19 | F | Hamartoma | U/K | U/K | U/K | 205 mm | 1 | Well | Autopsy | |
| 7 | Heinke | [11] | 39 | M | Hamartoma | U/K | U/K | U/K | 32 mm | 1 | Moderate | PHx | U/K |
| 8 | Kim | [12] | 74 | M | Hamartoma | No | No | No | 40 × 50 mm[a] | 1 | Well | PHx | > 2 years 6 months |
| 9 | Ijima | [13] | 29 | M | Caroli | No | Yes | No | 40 mm | 1 | Moderate | PHx | > 2 years 5 months |
| 10 | Kchir | [14] | 70 | F | Caroli | U/K | U/K | No | 45 mm | 1 | | PHx | 4 days |

*PH* portal hypertension, *U/K* unknown. When renal disease was present, "Yes" in the column of Kidney, *TACE* transcatheter arterial chemoembolization, *RFA* radiofrequency ablation, *PHx* partial hepatectomy, *N/A* not available

[a]Also had Cholangiocellular carcinoma (20 × 20 mm)

In conclusion, we successfully treated HCC arising from CHF using RFA under laparoscopy. This locoregional therapy is acceptable for the treatment of HCC arising from CHF in cases with no history of recurrent cholangitis and well-controlled portal hypertension.

## Compliance with ethical standards

**Conflict of interest** All authors have nothing conflict of interests in this study.

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