CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 44 (2018) 220–225

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Complete spontaneous necrosis of hepatocellular carcinoma accompanied by portal vein tumor thrombosis: A case report

Yuichi Goto [a,*], Yoshihiro Uchino [a], Shin Sasaki [a], Nobuhisa Shirahama [a], Yoriko Nomura [a], Jun Akiba [b], Hiroto Ishikawa [a], Yoshito Akagi [a], Hiroyuki Tanaka [a], Koji Okuda [a]

[a] Division of Hepatobiliary and Pancreatic Surgery, Department of Surgery, Kurume University School of Medicine, 67 Asahi-machi, Kurume, 8300011, Japan
[b] Department of Pathology, Kurume University School of Medicine, 67 Asahi-machi, Kurume, 8300011, Japan

ARTICLE INFO

Article history:
Received 17 January 2018
Received in revised form 21 February 2018
Accepted 28 February 2018
Available online 7 March 2018

Keywords:
Complete spontaneous necrosis
Hepatocellular carcinoma
Portal vein tumor thrombosis
Spontaneous regression
Portal vein ligation

ABSTRACT

INTRODUCTION: We report a rare case of complete spontaneous necrosis of a hepatocellular carcinoma (HCC) accompanied by portal vein tumor thrombosis (PVTT), as confirmed by resection.
CASE PRESENTATION: A 64-year-old man was referred to our hospital for suspected HCC. Contrast-enhanced computed tomography (CECT) findings before admission revealed a 53-mm tumor in the posterior segment of the liver and were suspicious for PVTT in the right posterior PV. Both alpha-fetoprotein (AFP) and proteins induced by vitamin K absence or antagonist-II (PIVKA-II) were elevated at 17,562 ng/mL and 153 mAU/mL, respectively. We diagnosed the findings as HCC with PVTT. Seven days after the first CECT scan, we performed CECT volumetry, which revealed that the tumor had regressed to 30 mm, along with regression of the PVTT. We performed portal vein ligation (PVL), and 10 days later, CECT revealed that the tumor had shrunk to 20 mm. AFP and PIVKA-II levels were 643 ng/mL and 14 mAU/mL, respectively. We suspected spontaneous regression of the patient's HCC, but performed a hepatectomy. Histopathology revealed a 22-mm tumor with a thin fibrous capsule and a tumor thrombus in the PV. Trabecular and pseudoglandular structures consisting of denucleated HCC epithelial cells made up both the tumor and thrombus, and the finding confirmed the spontaneous necrosis of HCC.
CONCLUSIONS: We present an extremely rare case of complete spontaneous necrosis of HCC with PVTT. When spontaneous necrosis is suspected, surgery should be considered because of the potential risk of residual viable cancer cells.

1. Introduction

Spontaneous massive necrosis of hepatocellular carcinoma (HCC) is observed about 2% of patients, especially those with large tumors; whereas spontaneous complete necrosis is extremely rare, with 1 case occurring per 6000–10,000 cases [1]. Only 17 English-language reports of spontaneous complete necrosis of HCC as confirmed by resected specimens were published between 1987 and 2017 [1–17]. Of these reports, only 1 described complete spontaneous necrosis of HCC with gross portal vein tumor thrombosis (PVTT) [10]. Herein, we present an extremely rare case of complete spontaneous necrosis of HCC with PVTT. This paper has been reported in line with the SCARE criteria [18].

2. Case presentation

A 64-year-old man with a liver tumor detected by abdominal ultrasonography (US) was referred to our hospital. The patient's past history included hepatitis C virus infection treated by interferon therapy 9 years previously. He achieved sustained virological response. The patient was negative for a history of alcohol consumption; smoking; excessive weight loss; and medications, including herbs. The patient's height and weight were 1.68 m and 52.6 kg, respectively with no specific physical abnormalities. Contrast-enhanced computed tomography (CECT) before admission revealed a 53-mm tumor in hepatic segments 6 and 7, and findings suggestive of PVTT in the right posterior portal vein (PV) (Fig. 1). The tumor and suspected PVTT were slightly enhanced during the early-phase CECT (Fig. 1A) and were washed out dur-

Abbreviations: AFP, alpha-fetoprotein; AFP-L3, *Lens culinaris* agglutinin-reactive AFP isoform 3; CECT, contrast-enhanced computed tomography; HCC, hepatocellular carcinomaPIVKA-II – proteins induced by vitamin K absence or antagonist-II; PV, portal vein; PVL, portal vein ligation; PVTT, portal vein tumor thrombosis; US, ultrasonography.

* Corresponding author.
E-mail addresses: gotou_yuuichi@med.kurume-u.ac.jp (Y. Goto), yos205dr@yahoo.co.jp (Y. Uchino), sasaki_shin@med.kurume-u.ac.jp (S. Sasaki), shirahama_nobuhisa@med.kurume-u.ac.jp (N. Shirahama), nomura_yoriko@med.kurume-u.ac.jp (Y. Nomura), akiba@med.kurume-u.ac.jp (J. Akiba), campxxx@med.kurume-u.ac.jp (H. Ishikawa), yoshisg@med.kurume-u.ac.jp (Y. Akagi), htanaka@med.kurume-u.ac.jp (H. Tanaka), kook@med.kurume-u.ac.jp (K. Okuda).

https://doi.org/10.1016/j.ijscr.2018.02.045

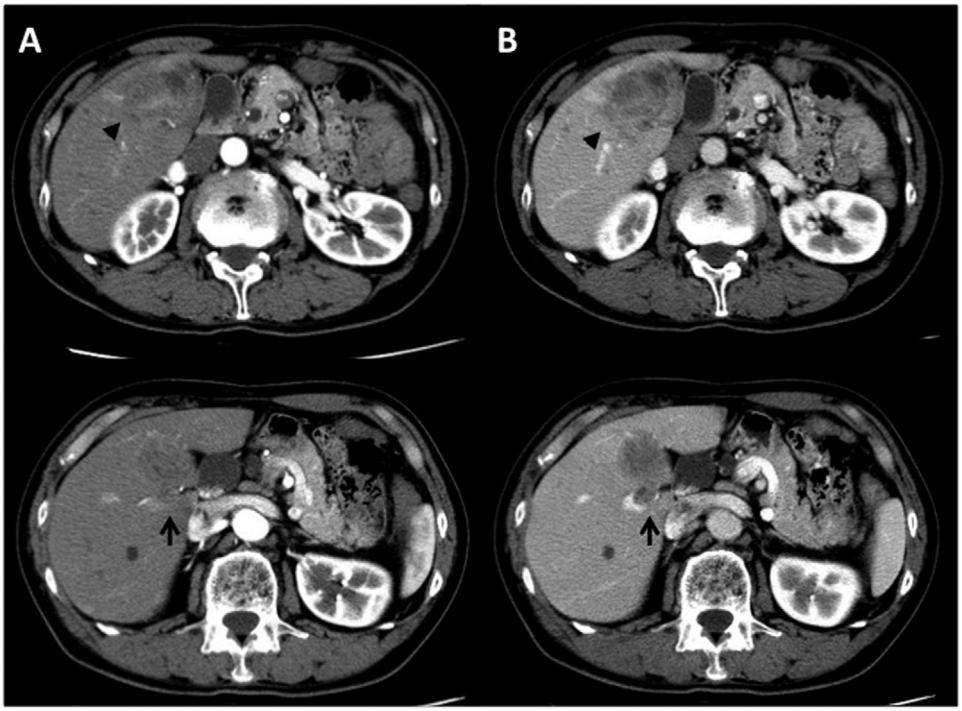

Fig. 1. Contrast-enhanced computed tomography (CECT) scan for initial diagnosis. The image shows a tumor 53 mm in size (arrowhead) and portal vein tumor thrombosis (PVTT) in the posterior branch of the portal vein (arrow). The tumor and suspected PVTT are slightly enhanced during the early-phase CECT (A) and are washed out during the equilibrium phase (B).

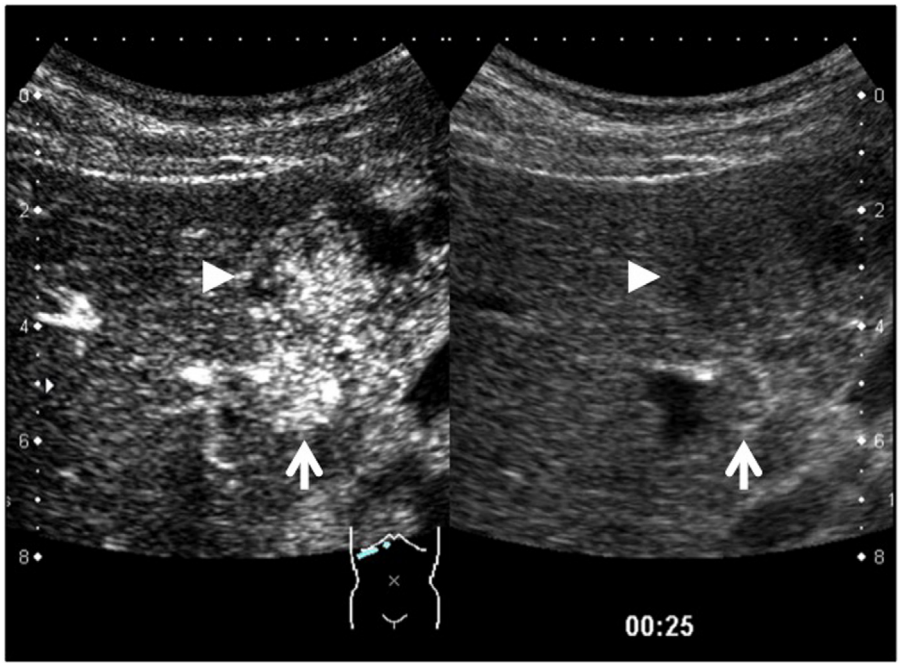

Fig. 2. Arterial phase of contrast-enhanced ultrasonography at diagnosis. Main tumor (arrowhead) and suspected portal vein tumor thrombosis (arrow) are well enhanced.

ing the equilibrium phase (Fig. 1B). However, both the tumor and suspected PVTT were well enhanced during the arterial phase and washed out in the postvascular phase on contrast-enhanced US (Fig. 2). Upper gastrointestinal endoscopy and colonoscopy revealed nonspecific findings. The results of preoperative laboratory testing were as follows: white blood cell count, 5300 cells/μL; red blood cell count, 463×10^4 cells/μL; serum hemoglobin concentration, 14.6 g/dL; serum platelet count, 19.5×10^4 platelets/μL; serum aspartate aminotransferase, 12 IU/L; serum alanine aminotransferase, 16 IU/L; serum alkaline phosphatase, 229 IU/L; serum gamma glutamic transpeptidase, 29 IU/L; total serum bilirubin, 1.2 mg/dL; serum albumin, 4.58 g/dL; serum C-reactive protein, <0.04 mg/dL; prothrombin time (%), 88%; hemoglobin A1c, 5.7%; indocyanin green retention rate after 15 min, 26.0%. The levels of serum alpha-fetoprotein (AFP) and proteins induced by vitamin K absence or antagonist-II (PIVKA-II) were elevated at 17,562 ng/mL and 153 mAU/mL, respectively, with a percentage of the *Lens culinaris* agglutinin-reactive AFP isoform 3 (AFP-L3) of