A female outpatient in her 50s had routinely visited our hospital because of liver cirrhosis resulting from hepatitis B virus and biliary duct stones.
At 65 years of age, she suffered from HCC with BCLC early stage A.
Her liver function was well preserved, and Child-Pugh score was A.
She underwent left lateral segmentectomy for HCC, without other preoperative treatments.
Histopathological findings revealed bridging fibrosis and pseudolobule formation.
Serum levels of alpha-fetoprotein (AFP) and protein induced by vitamin K absence-II normalized after primary resection (Figure 1).
In imaging studies, no LN metastasis was detected at the time of primary resection of HCC.
Two and a half years postoperatively, the patient's AFP level increased dramatically to 780.2 ng/ml.
A lobular lesion with fine enhancement was detected by contrast-enhanced magnetic resonance imaging.
The tumor measured 27 mm in diameter and was located caudally on the left kidney.
In contrast-enhanced computed tomography, the tumor showed strong enhancement in the arterial phase (Figure 2A, 2B) and a relatively low density in the portal phase (Figure 2C, 2D).
These enhancement findings appeared consistent with a typical HCC pattern.
Detailed imaging studies of both magnetic resonance imaging and computed tomography were performed in this case because a very rare metastatic LN initially seemed to be debatable and we needed to rule out exclusion diagnoses.
Three-dimensional imaging proved that the tumor was fed by a main vessel from the inferior mesenteric artery (red arrow) and by an accessory feeder from the superior mesenteric artery (Figure 3).
Fluorine-18-fluorodeoxyglucose positron emission tomography (FDG-PET) and positron emission tomography-computed tomography did not detect the tumor (Figure 4), although we thought positron emission tomography-computed tomography was helpful to identify other metastatic tumors.
Further imaging findings revealed no other intrahepatic or extrahepatic metastasis.
Based on the tumor location, the clinical diagnosis was solitary metastasis to a mesocolic LN or HCC dissemination.
Determining the ideal therapeutic strategy for solitary but extrahepatic rare metastasis was difficult.
Although rapid growth was a critical concern in this case, the tumor was solitary and not accompanied by other metastases.
Considering both diagnostic and therapeutic viewpoints, we finally chose surgical resection in this case.
No disseminative nodules, lymphadenopathy, or ascites was observed during surgery.
The tumor was located in the mesocolon nearly at the wall of the descending colon, and partial resection of the descending colon with regional mesocolon was performed.
The patient's postoperative course was uneventful, and she was discharged on postoperative day 8.
Serum levels of tumor marker decreased immediately after surgery (Figure 1).
Macroscopically, the mesocolic tumor was a solid and elastic mass with a smooth surface (Figure 5A).
A yellowish nodule was encapsulated in the cut surface (Figure 5B).
The enlarged LN contained metastatic HCC with a ductal structure (Figure 6A), and immunohistochemically, the tumor was positive for AFP and negative for CK-20, which was consistent with the pattern of primary HCC (Figure 6B).
The histopathological diagnosis was metastatic HCC to a mesocolic LN.
As of the writing of this report, the patient has remained free of recurrence for 13 months after the second surgery, and has also been carefully followed up.
No adjuvant therapies have been performed.