A Large Carcinosarcoma of the Gallbladder Accompanied by Pancreatobiliary Maljunction - A Case with a Six-year Survival

Hiroyuki Matsubayashi¹, Toru Matsui¹, Teichi Sugiura², Rie Makuuchi³, Junichi Kaneko¹, Junya Satoh¹, Tatsunori Satoh¹, Shinya Fujie¹, Hirotoshi Ishiwatari¹, Keiko Sasaki⁴ and Hiroyuki Ono¹

Abstract:

Pancreatobiliary maljunction (PBM) is a rare congenital malformation, often associated with adenocarcinoma. However, PBM accompanying gallbladder carcinosarcoma has rarely been reported. A 72-year-old woman was referred to our hospital, complaining of abdominal pain. Computed tomography showed a polypoid mass in the gallbladder. Endoscopic retrograde cholangiopancreatography showed PBM, and aspirated bile demonstrated elevated levels of pancreatic-type amylase (26,780 U/L) and cancer cells. Extended cholecystectomy was performed. Histologically, the tumor had adenocarcinoma, squamous cell carcinoma and sarcoma components. Despite the large tumor size (84 mm) and intra-vessel cancer permeations, this patient has been healthy for 73 months since the surgery.

Key words: carcinosarcoma, gallbladder, pancreatobiliary maljunction, prognosis


Introduction

Carcinosarcoma is a malignant tumor composed of both carcinomatous and sarcomatous elements (1). This histological type of tumor can develop in all types of organs (2-5), but its occurrence in the gallbladder is quite rare, accounting for less than 1% of all gallbladder malignancies (6).

Biliary cancer can occur in response to pancreatobiliary maljunction (PBM), a congenital malformation. In PBM, the pancreatobiliary duct union occurs outside the duodenal wall, and this anatomic anomaly causes continuous and chronic exposure of refluxed pancreatic juice to the biliary epithelium. The histology of these PBM-related biliary cancers is almost always adenocarcinoma, as most of these cancers (39%-91%) develop in the background of biliary epithelial hyperplasia (7, 8). The anatomic pattern shows a correlation with the cancer location, as the incidence of bile duct cancer is greater in cases with congenital biliary dilatation (32%) than in those without this congenital anomaly (7%). By contrast, gallbladder cancer is less frequent in cases with congenital biliary dilatation (62%) than in those without it (88%) (9).

Gallbladder cancer accompanying PBM is now being increasingly frequently reported; however, carcinosarcoma of the gallbladder accompanying PBM has seldom been reported in the English literature (10, 11). We herein report a case with a six-year post-operative survival in a patient diagnosed with gallbladder carcinosarcoma accompanied by PBM.

Case Report

A 72-year-old woman visited her nearest hospital complaining of nausea and abdominal pain in her right upper quadrant. Abdominal ultrasonography (US) (Fig. 1a) showed
Laboratory data showed elevated levels of serum alkaline phosphatase (ALP; 459 IU/L) and gamma-glutamyl transpeptidase (γ-GTP; 111 IU/L); other measurements, including those of tumor markers (carcinoembryonic antigen: 2.3 ng/mL, normal range: ≤5.0 ng/mL, and carbohydrate antigen 19-9: 15 U/mL, normal range: ≤37 U/mL), were normal.

Enhanced US revealed heterogeneous and strong contrast enhancement within the tumor from 10 seconds until 3 minutes after contrast injection (Fig. 1b), with diminished enhancement afterward. Multi-detector computed tomography (CT) (Fig. 2a) showed a large, irregularly shaped polypoid mass (48×16 mm) with heterogeneous wall thickness in the gallbladder. 18F-fluorodeoxyglucose-positron emission tomography (FDG-PET) showed a strong uptake at the gallbladder (SUV max: 13.64) (Fig. 3). Magnetic resonance imaging (MRI) demonstrated heterogeneously low-intensity signals within the tumor on T1-weighted imaging, high-intensity signals on T2-weighted imaging, and reduced diffusing capacity on diffusion-weighted imaging (Fig. 4a-4c).

Figure 1. Abdominal ultrasonography. A large polypoid lesion is recognized in the gallbladder (a). The tumor was diffusely and strongly enhanced by microbubble contrast (b).

Figure 2. Enhanced computed tomography (CT). A hypervascular polypoid lesion evident within the gallbladder (a) progressed and invaded the liver within six weeks (b).

Figure 3. 18F-fluorodeoxyglucose-positron emission tomography (FDG-PET). A strong uptake is seen at the gallbladder.
Magnetic resonance cholangiopancreatography (MRCP) was suggestive of PBM (Fig. 4d), and this diagnosis was confirmed by endoscopic retrograde cholangiopancreatography (ERCP) (Fig. 5).

Bile juice aspirated from the common bile duct demonstrated a high level of pancreatic-type amylase (26,780 U/L), and the presence of cancer cells was confirmed by cytology. Multiple stepwise forceps biopsies obtained from the hilar common duct and the superior, middle and inferior sites of the common bile duct all revealed non-neoplastic biliary epithelia. Extended cholecystectomy was scheduled based on the diagnosis of gallbladder cancer (GBC) associated with PBM; however, the patient refused surgery at that time.

Forty-five days after the initial diagnosis, she revisited our hospital with appetite loss. Repeat CT demonstrated considerable growth of the gallbladder tumor (90×85 mm) and apparent spread to the liver (Fig. 2b), so the surgery was performed 2 weeks later. On laparotomy, the hepatic invasion of the tumor was found to be less extensive than anticipated;
Therefore, extended cholecystectomy was conducted without hepatic segmentectomy or lobectomy.

Regarding its gross appearance, the gallbladder tumor measured 84×72 mm in size, appeared rugged, and was attached to the liver bed. A cut section revealed that the entire cavity had been replaced by a yellowish solid tumor with bleeding necrosis (Fig. 6a). Histologically, the tumor consisted of three components (adenocarcinoma, squamous cell carcinoma and sarcoma) showing an intermediate growth pattern (INFb) with scanty stroma (medullary type) (12). The sarcoma component consisted largely of polymorphic cells and bundles of spindle cells, and this component occupied a large part of the tumor in the contiguous liver bed (pHinf1b) (12). Transition among the three histological components was recognized, and a diagnosis of so-called carcinosarcoma was made.

Immunohistochemical staining of the adenocarcinoma component was positive for cytokeratin but negative for vimentin, whereas the sarcoma component staining was positive for vimentin but negative for cytokeratin (Fig. 6b-6e). Immunostaining of TP53 was diffusely overexpressed, and the Ki-67 labeling index was 60%-80% in...
the tumor. Invasion to the lymph vessel and peripheral vein was noted, but neural invasion was not seen. The surgical margin was negative for cancer, and lymph node metastasis was also negative (Stage IIIA by Japanese classification) (12). The patient’s postoperative course was uneventful, and she was discharged 16 days after the operation. At 73 months after the surgery, she remained alive with no evidence of recurrence.

Discussion

Carcinosarcoma of the gallbladder (CSGB) is a rare neoplasm. However, according to our literature survey of PubMed and the Japan Medical Abstracts Society, more than 100 cases have been reported in the English and Japanese literature.

The findings of 35 of the Japanese cases reported in the last 15 years (2004-2018) are summarized in Table (13-44). Including our case, the mean age was 72 years old, showing a female predominance (13 men and 23 women). They were diagnosed mostly with a complaint of abdominal pain and showed a large tumor size (mean: 65 mm, range: 16-120 mm). Three CSGB cases accompanied by PBM were noted among these Japanese reports (Table), in addition to two cases reported in the English literature (10, 11).

Our patient also demonstrated a PBM. PBM is a well-known risk factor for gallbladder cancers (45), as the reflux of pancreatic juice into the biliary tract induces epithelial changes (hyperplasia) associated with long-term inflammation, which eventually lead to carcinogenesis (46). A Japanese nationwide survey reported that, among adult patients with congenital biliary dilation, 6.9% and 13.4% had cancers of the bile duct and gallbladder, respectively. In cases with PBM without biliary dilation, the rates of cancers of the bile duct and gallbladder were 3.1% and 37.4%, respectively (47). In our case, the common bile duct was slightly dilated (14 mm), but cancer of this area was clinically excluded by multiple stepwise biopsies before surgery. Nevertheless, the risk for developing cancer in the remnant biliary tract is still high, so careful follow-up is needed for this patient in the future.

CSGB is classified into two categories: true carcinosarcoma and so-called carcinosarcoma. True carcinosarcoma is diagnosed histologically, based on differentiation of the mesenchymal element into neoplastic bone and osteoid (26, 48, 49). The so-called carcinosarcoma is diagnosed when a spindle cell carcinoma (the sarcomatous component) originates from the dedifferentiated adenocarcinoma component; therefore, a histologically confirmed transitional finding is a key feature. The present case showed a transition of two elements, but no bone, osteoid or rhabdoid elements were observed. Immunohistochemistry showed cytokeratin staining mainly in the carcinomatous component, whereas vimentin staining was mainly confined to the sarcomatous area. Thus, the present case was diagnosed as a “so-called carcinosarcoma of the gallbladder” (11).

The preoperative diagnosis of CSGB is difficult because of the lack of radiological findings or serum markers specific for this entity (26). In the previous Japanese cases, serum CEA levels were within the normal limits or faintly elevated, and CA19-9 levels were markedly elevated only in a small fraction (>100 U/mL: 14.8% [4/27]) (Table). A typical CSGB tends to grow intraluminally with a polypoid form rather than by infiltration to adjacent organs (50) (Table). Nevertheless, 15%-25% of adenocarcinomas of the gallbladder progress similarly to a macroscopic polypoid lesion. In the present case, the initial appearance was polypoid, and the tumor seemed to be noninvasive; however, it grew rapidly within a short period similar to the other reported cases (cases 18 and 25 in Table). Based on the tumor size, extended cholecystectomy was performed. Despite the aggressive behavior shown in the sequential images, the pathology of the tumor showed an expansive rather than invasive growth, and the liver invasion was limited to a few millimeters. This discrepancy may reflect the growth pattern typically shown by sarcoma cells, which is expansive rather than the invasive type common to ordinary gallbladder adenocarcinomas (26, 48, 49). Consequently, the tumor was removed en bloc, and R0 resection was achieved.

Most gallbladder cancer patients present with advanced-stage disease (51, 52). The prognosis of patients with serosal or liver invasion is especially poor, and the surgical outcomes are not always sufficient to confer any long-term survival benefit (1, 53). The survival of CSGB patients is also generally poor (54). A review by Zhang et al. of 68 cases of CSGB indicated a median survival time of 5 months, a 1-year survival rate of 19.5% and a 5-year survival rate of 16.5% (55). However, in cases where curative resection was performed for carcinosarcomas with invasion limited to the muscularis propria, the 5-year survival rate increased to 88.9% (56). Among Japanese cases (Table), a similar trend was recognized, and the post-operative prognosis was significantly longer in stage I-III cases than in stage IV cases (1-year survival rate: 86.7% vs. 37.5%, p=0.03, 5-year survival rate: 75.0% vs. 14.3%, p=0.04 by Fisher’s test). The radical operation performed in the present case was considered to be one reason for the patient’s favorable outcome (73 months of survival without recurrence). Therefore, for patients with gallbladder CSGB, surgical resection in the early stage is essential for a positive long-term prognosis.

Conclusion

Differentiating CSGB from ordinary GBC is difficult because of their overlapping imaging features. Some CSGBs demonstrate an intraluminal growth pattern, but these lesions may be able to be cured by radical surgery when the tumor invasion is limited. Careful surveillance is needed for biliary tract malignancies in patients with pancreaticobiliary malformations.

The authors state that they have no Conflict of Interest (COI).
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<td>Tonochoi</td>
<td>40</td>
<td>2015</td>
<td>87</td>
<td>M</td>
<td>abdominal pain</td>
<td>ND</td>
<td>ND</td>
<td>60</td>
<td>s (colon) low mass</td>
<td>GBC</td>
<td>so-called ND</td>
<td>C, TC</td>
<td>D</td>
<td>**</td>
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<tr>
<td>32</td>
<td>Karahashi</td>
<td>41</td>
<td>2015</td>
<td>64</td>
<td>M</td>
<td>hematemesis</td>
<td>ND</td>
<td>ND</td>
<td>100</td>
<td>s (du, colon) irregular high node</td>
<td>GBC</td>
<td>so-called IVA</td>
<td>HPD (S6) → S-1 → GEM-Pclatin</td>
<td>A</td>
<td>17m (rec)</td>
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<tr>
<td>33</td>
<td>Yoneyama</td>
<td>42</td>
<td>2015</td>
<td>85</td>
<td>F</td>
<td>right-hypochondrialgia nausea, fatigue</td>
<td>ND</td>
<td>95.5</td>
<td>50</td>
<td>s(liver) high polypoid</td>
<td>GBC</td>
<td>ND</td>
<td>IVA</td>
<td>C, TC</td>
<td>A</td>
<td>7y</td>
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<tr>
<td>34</td>
<td>Nagatsu</td>
<td>43</td>
<td>2015</td>
<td>69</td>
<td>F</td>
<td>nausea, fatigue</td>
<td>ND</td>
<td>ND</td>
<td>70</td>
<td>s(liver, du) irregular high mass</td>
<td>GBT</td>
<td>so-called IIA</td>
<td>HPD → R</td>
<td>A</td>
<td>5m (rec)</td>
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<tr>
<td>35</td>
<td>Endoh</td>
<td>44</td>
<td>2016</td>
<td>70s</td>
<td>F</td>
<td>upper abdominal pain</td>
<td>2.4</td>
<td>255.8 (+) 50</td>
<td>ss</td>
<td>irregular high mass</td>
<td>GBC</td>
<td>so-called II</td>
<td>C → S1+GEM, PH → GEM</td>
<td>A</td>
<td>32m (rec)</td>
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<tr>
<td>36</td>
<td>Present case</td>
<td>2019</td>
<td>72</td>
<td>F</td>
<td>abdominal pain</td>
<td>2.4</td>
<td>15 (+) 48 →90 (1.5m)</td>
<td>s(liver) high irregular high</td>
<td>GBC</td>
<td>so-called IIA</td>
<td>ERH, EHBDR</td>
<td>A</td>
<td>73m</td>
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</table>

#tumor originated from gallbladder, liver or omentum, *tumors were incidentally detected by image examinations, **died early post-operative days.


Authors’ contribution: H.M. and T.M. performed endoscopic procedures, managed the patient and wrote the manuscript. R.M. and T.S. performed the surgical management. K.S. made the pathological diagnosis. J.K., J.S., T.S., S.F., H.I., and H.O. provided beneficial comments. H.M. and T.M. contributed equally to this case report.

References


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