Pure pancreatic hepatoid carcinoma: a surgical case report and literature review

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Abstract

Background: Hepatoid carcinoma (HC) is an extra-hepatic neoplasm that shares the morphological and immunohistochemical features of hepatocellular carcinoma. Pancreatic HC exists as either pure or combined type. Pure pancreatic HC is extremely rare, with only a few cases reported in the literature to date. Because of the rarity of pure pancreatic HC, its clinical features including incidence, behavior, and prognosis remain unclear. We herein report the case of a 56-year-old man who developed pure pancreatic HC treated with surgical resection. We also include a review of the existing literature.

Case presentation: A 56-year-old male patient was admitted to our hospital after a pancreatic cyst was identified by abdominal ultrasonography on a comprehensive medical examination. Endoscopic ultrasound revealed a cystic mass measuring 13 mm in size in the pancreatic head and a low-density mass measuring 16 mm in size in the pancreatic tail, which was partially enhanced on contrast-enhanced ultrasound. Contrast-enhanced computed tomography (CT) revealed a branch duct type intraductal papillary mucinous neoplasm in the pancreatic head and an early enhanced nodule measuring approximately 10 mm in size in the pancreatic tail. Endoscopic ultrasound-guided fine-needle aspiration of the hypervascular tumor was performed. The hypervascular tumor was suspected to be a solid pseudopapillary neoplasm. Laparoscopic spleen-preserving distal pancreatectomy was performed. Histology was identical to hepatocellular carcinoma of the liver. Immunohistochemically, the tumor cells were positive for hepatocyte paraffin 1, and a canalicular pattern was confirmed on the polyclonal carcinoembryonic antigen staining. The patient was diagnosed with a moderately differentiated pancreatic HC. The patient was followed up without adjuvant chemotherapy, and there was no evidence of recurrence at 6 months post-operatively.

Conclusions: We present a case of moderately differentiated pure pancreatic HC. For the accurate preoperative diagnosis of pure pancreatic HC, biopsy is preferred to cytology or preoperative imaging studies such as CT. The prognosis of pure pancreatic HC depends on its differentiation.

Keywords: Hepatoid carcinoma, Pancreatic cancer, Laparoscopic distal pancreatectomy, Hepatocyte paraffin 1, Polyclonal carcinoembryonic antigen
Background
Hepatoid carcinoma (HC) is an extra-hepatic neoplasm that shares the morphological and immunohistochemical features of hepatocellular carcinoma. The first case of HC reported by Ishikura in 1985 was of a gastric neoplasm with these characteristics [1]. HC is a rare neoplasm that has been described in different organs such as the stomach, pancreas, lung, gallbladder, ovary, and colon [2–7]. With the most common location being the stomach, followed by the ovaries [8–13].

Several cases of pancreatic HC have been reported since pancreatic HC was first reported by Yano in 1999 [14]. Pancreatic HC exists as pure type or combined type. Pure pancreatic HC has been referred to as hepatoid adenocarcinoma, hepatoid carcinoma, and ectopic hepatocellular carcinoma [14–16]. Combined pancreatic HC refers to HC with other histological components such as neuroendocrine tumor, endocrine carcinoma, islet cell glucagonoma, or pancreatic ductal adenocarcinoma [17–19]. Pure pancreatic HC is extremely rare, with few cases having been reported to date in the English literature. Because of the rarity of pure pancreatic HC, its clinical features including the incidence, behavior, and prognosis remain unclear.

We herein report the case of a 56-year-old man who developed pure pancreatic HC and was treated with surgical resection. We also conducted a review of the previous literature.

Case presentation
A 56-year-old male patient presented with a pancreatic cyst identified by abdominal ultrasonography on a comprehensive medical examination and was admitted to our hospital. He had a past medical history of type 2 diabetes, hyperlipidemia, and chronic hepatitis C for which he received interferon therapy for chronic hepatitis C more than 20 years previously. He had no family history of cancer. Laboratory tests revealed normal levels of carcinoembryonic antigen (CEA) and carbohydrate antigen 19–9, and alpha-fetoprotein (AFP) and protein induced by vitamin K absence or antagonist-II (PIVKA-II) were absent. Endoscopic ultrasound (EUS) showed a cystic mass measuring 13 mm in size in the pancreatic head and a low-density mass measuring 16 mm in size in the pancreatic tail (Fig. 1a), which was partially enhanced on the contrast-enhanced ultrasound image (Fig. 1b). Contrast-enhanced computed tomography (CT) revealed branch duct type intraductal papillary mucinous neoplasms in the pancreatic head and an early enhanced nodule measuring approximately 10 mm in size in the pancreatic tail (Fig. 2a–c). An enhancement of the nodule lasted until the late phase, although its density was gradually attenuated. Magnetic resonance imaging (MRI) did not detect the corresponding nodule in the pancreatic tail.

Fig. 1 Endoscopic ultrasound findings. a Endoscopic ultrasound showed a low-density mass measuring about 16 mm in size in the pancreatic tail (circle). b The low-density mass in the pancreatic tail was partially enhanced on the contrast-enhanced ultrasound with bolus administration of Sonazoid.

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Based on the above features, our initial differential was that of a neuroendocrine tumor or a solid pseudopapillary neoplasm (SPN). Endoscopic ultrasound-guided fine-needle aspiration (FNA) was performed to make a definitive diagnosis. FNA cytology showed that the tumor cells exhibited an acidophilic cytoplasm with small, round nuclei. Immunohistochemistry was performed to differentiate between a neuroendocrine tumor, SPN, and acinar cell carcinoma. The tumor cells were positive for cytokeratin, nuclear/membranous β-catenin, CD10, and CD56 and were negative for chromogranin A, synaptophysin, progesterone receptor, vimentin, and Bcl-10. Therefore, we suspected that the hypervascular tumor in the pancreatic tail was suspected to be SPN, but the results were not convincing. Laparoscopic spleen-preserving distal pancreatectomy was performed. Macroscopically, a well-circumscribed whitish-yellow solid mass, measuring 7 mm in the greatest dimension, was found in the pancreatic tail (Fig. 3). Histologically, polygonal tumor cells with round nuclei and abundant eosinophilic cytoplasm formed thick trabeculae. The differentiation was moderate (Fig. 4). Immunohistochemically, the tumor cells were positive for hepatocyte paraffin 1, AE1/AE3, and CD10 and negative for AFP, progesterone receptor, vimentin, chromogranin A, and synaptophysin (Fig. 5a). A canalicular pattern was confirmed on the polyclonal CEA staining (Fig. 5b). HC is characteristically hepatocyte paraffin 1 (HepPar1)-positive and has a canalicular pattern on polyclonal CEA staining. Finally, a diagnosis of moderately differentiated pancreatic HC was made. The patient’s postoperative course was uneventful, and he was discharged in good health 10 days after the operation. The patient did not receive adjuvant chemotherapy and remained recurrence-free at 6 months after the surgery. The serum levels of AFP (3 ng/mL) and PIVKA-II (28 mAU/mL) were normal at 1 month after the surgery. The latest serum levels of AFP (2 ng/mL) and PIVKA-II (25 mAU/mL) were normal at 6 months after the surgery.

**Discussion**

Histologically, pancreatic HC can be categorized as either pure type or combined type. We conducted a systematic review of the English literature using the PubMed search engine and found 20 resected cases of pure pancreatic HC. The present case and 20 reported cases of resected pure pancreatic HC are summarized in Table 1 [28–32, 34–37].

The median age of patients with resected pure pancreatic HC was 59 years (range 32–83), and most patients were men (81%). With regard to tumor location, most
tumors (81%) were located in the body or tail of the pancreas, whereas approximately 75% of all pancreatic carcinomas occurred in the head or neck of the pancreas [20]. We found that 47% of the patients who had resectable pure pancreatic HC in the body or tail of the pancreas had no symptoms at the time of medical examination. Conversely, 75% of the patients with resectable pure pancreatic HC in the head of the pancreas had some symptoms. Clinically, the patients who had resectable pure pancreatic HC in the body or tail of the pancreas were less symptomatic than those with tumors located in the head of the pancreas.

The diagnosis of pure pancreatic HC based solely on preoperative imaging studies is difficult due to the non-specific radiologic features of this tumor [21]. In fact, none of the patients who underwent preoperative imaging studies such as CT received a precise diagnosis because of the radiographic characteristics with various vascular patterns in tumor enhancement. Therefore, preoperative imaging studies alone are insufficient to accurately diagnose pure pancreatic HC.

Immunohistochemical studies play an important role in making a definitive diagnosis. However, biopsy provides a more accurate diagnosis than cytology. In a series of 21 cases of resected pure pancreatic HC, a biopsy was performed in 5 cases and cytology was performed in 7 cases for preoperative workup. In the biopsy cases, 2 of 5 cases (40%) were positive for HepPar1 on immunohistochemical staining and were diagnosed with pancreatic HC. Conversely, 5 of 7 cytology cases (71%) were misdiagnosed as other cancers such as SPN. Considering that the overall 5-year survival rate of SPN is approximately 95% [22], and that of pancreatic HC is 40.4% [23], an accurate diagnosis of pancreatic HC is very important. This suggests that biopsy is essential for the preoperative diagnosis of pure pancreatic HC. In the present case, preoperative immunohistochemistry precluded correct diagnosis because of the small amount of the tissue and the under-recognition of this tumor.

While many hepatocellular carcinoma tumors express AFP and PIVKA-II at levels which may be detectable in the serum, preoperative AFP and PIVKA-II are not usually measured in patients with pancreatic HC. The rarity of this tumor makes it difficult to endorse the sensitivity of AFP and PIVKA-II as a screening test for pure pancreatic HC. Nevertheless, these may be useful markers for postoperative surveillance.

![Fig. 4](image1.jpg)

**Fig. 4** Hematoxylin and eosin staining findings. Polygonal tumor cells with round nuclei and abundant eosinophilic cytoplasm formed thick trabeculae in the tumor. The differentiation was moderate.

![Fig. 5](image2.jpg)

**Fig. 5** Immunohistochemical staining findings. 
(A) Hep Par1
(B) Polyclonal CEA
Previous reports have described that pancreatic HC usually has an aggressive clinical course and an extremely poor prognosis \[11, 24\]. Recently, Yang et al. \[25\] reported the prognosis of pancreatic HC. The authors described four histological subtypes of pancreatic HC, namely, with (1) pure HCC-like morphology, (2) neuroendocrine differentiation, (2) true glandular differentiation, and (4) acinar cell differentiation. Pure pancreatic HC was associated with better disease-specific survival than the other subtypes. The 5-year disease-

| Table 1 | Reported surgical cases of pure pancreatic hepatoid carcinoma in the literature |
|---------|--------------------------------------------------------------------------------|
| Reference | Age | Gender | Location | Symptoms | Preoperative imaging study | Preoperative diagnostic method | Preoperative diagnosis | Postoperative pathological differentiation | Recurrence | Outcome |
| [14] | 57 M | Head | Epigastric pain, vomiting, and fever | CT | None | Not listed | Well to moderate | Liver metastasis | Dead, 2.8 months |
| [28] | 70 M | Body | None | CT | None | Not listed | Well | None | Alive, 12 months |
| [16] | 51 M | Body and tail | Upper gastrointestinal hemorrhage | CT, EUS | Biopsy | Nondiagnostic tissue | Moderate | None | Alive, 14 months |
| [29] | 32 M | Tail | Epigastric pain | CT | Biopsy | Hepatoid carcinoma | Well | None | Alive, 18 months |
| [26] | 49 F | Tail | Body weight loss | US, CT, MRI, ERCP | Pancreatic juice cytology | Adenocarcinomatous cells | Well | Liver metastasis | Alive, 48 months |
| [15] | 58 M | Body | Back and flank pain | CT | FNA | Hepatocytes | Well | None | Alive, 15 months |
| [11] | 53 F | Body and tail | Epigastric pain | Not listed | None | Not listed | Moderate | Liver metastasis | Alive, 22 months |
| [12] | 79 F | Body | None | CT | None | Not listed | Poor | Liver metastasis | Dead, 2 months |
| [30] | 80 M | Head | Nausea, emesis, diarrhea, and weight loss | MRI | None | Not listed | Moderate | None | Alive, 8 months |
| [31] | 69 M | Body and tail | Chest pain | CT, EUS | FNA | Hepatoid carcinoma | Moderate | None | Alive, 4 months |
| [32] | 61 F | Tail | None | CT, PET-CT, EUS | FNA | Lymphoma or neuroendocrine tumor | Moderate | None | Alive, 60 months |
| [33] | 57 M | Head | Jaundice | CT | None | Not listed | Moderate | None | Alive, 18 months |
| [34] | 47 M | Tail | Groin pain and backache | US, CT, MRI | None | Not listed | Moderate | None | Alive, 8 months |
| [21] | 59 M | Body | None | US, CT, MRI, EUS | FNA | SPN | Well | None | Alive, 12 months |
| [35] | 61 M | Tail | None | MRI, EUS | FNA | Acinar cell carcinoma | Moderate | None | Alive, 6 months |
| [23] | 67 M | Tail | None | CT, MRI | None | Not listed | Moderate | None | Alive, 6 months |
| [36] | 56 M | Tail | None | US, CT, MRI | None | Not listed | Moderate | None | Alive, 36 months |
| [37] | 78 M | Head | None | US, CT, MRI | Biopsy | Epithelial tumor | Well | None | Dead from acute heart attack, 2 months |
| [25] | 83 M | Body | Abdominal pain | CT | Biopsy | Neuroendocrine tumor | Moderate | None | Alive, 107 months |
| [25] | 72 M | Tail | Back pain | US, CT | None | Not listed | Well | None | Dead from pulmonary embolism, 1 month |
| Present case | 56 M | Tail | None | CT, MRI, EUS | FNA | SPN | Moderate | None | Alive, 6 months |

CT computed tomography, EUS endoscopic ultrasound, US ultrasound, MRI magnetic resonance imaging, ERCP endoscopic retrograde cholangiopancreatography, FNA fine needle aspiration, PET-CT positron emission tomography-computed tomography, SPN solid pseudopapillary neoplasm
specific survival rate of pure pancreatic HC was 77.3%, whereas that of pancreatic HC with neuroendocrine differentiation was 37.5% and that of pancreatic HC with true glandular differentiation or acinar cell differentiation was 0%. We re-reviewed the hematoxylin and eosin staining of 20 reported cases of resected pure pancreatic HC and assessed their differentiations. Among the resected cases, 7 cases were diagnosed as well-differentiated pure pancreatic HC, 13 cases were moderately differentiated, and 1 case was poorly differentiated. Excluding the sudden death cases, all the patients with well-differentiated pancreatic HC survived for more than 12 months after surgery. Contrarily, the poorly differentiated case had a short survival time of 2 months after surgery. Moreover, one patient with a poorly differentiated unresectable tumor who underwent chemotherapy (gemcitabine) died 3 months after the first consultation [9]. In brief, patients with poorly differentiated type tumors had a worse serious prognosis than did those with well or moderately differentiated type.

Recurrence after surgery was confirmed in 4 of 21 resected cases (19%), and in all cases, the liver was the site of recurrence [11, 12, 14, 26]. Among them, only one patient was reported to have received chemotherapy for the treatment of recurrent liver tumors. The 49-year-old female patient had a recurrence of liver metastasis 12 months after surgery and received chemotherapy with gemcitabine for an additional 26 months. She subsequently underwent right liver lobectomy 39 months after the initial operation because of an increase in tumor size [26]. As the frequency of postoperative liver metastasis recurrence is high, patients must be closely followed up after pancreatectomy. However, few studies have reported the optimal chemotherapy regimen for patients with pure pancreatic HC. We identified two patients with metastatic pancreatic HC treated with sorafenib, an oral multikinase inhibitor approved for the treatment of advanced hepatocellular carcinoma [10, 27]. In these reports, sorafenib seemed to provide some short-term clinical benefits. Therefore, sorafenib may be a possible candidate for chemotherapy in patients with recurrent or unresectable pure pancreatic HC.

Conclusions
We present a case of moderately differentiated pure pancreatic HC. Current clinical guidelines recommend biopsy as opposed to cytology or preoperative imaging studies for the preoperative diagnosis of pure pancreatic HC. While the natural history and prognosis of pure pancreatic HC may not be accurately predicted with these limited data, the prognosis of pure pancreatic HC could depend on the degree of differentiation.

Abbreviations
AFP: Alpha-fetoprotein; CEA: Carcinoembryonic antigen; CT: Computed tomography; EUS: Endoscopic ultrasound; FNA: Fine needle aspiration; HC: Hepatoid carcinoma; HepPar-1: Hepatocyte paraffin 1; MRI: Magnetic resonance imaging; PIVKA-II: Protein induced by vitamin K absence or antagonist-II; SPN: Solid pseudopapillary neoplasm

Acknowledgements
We would like to thank Editage (www.editage.jp) for English language editing.

Authors’ contributions
TT and MN conceived and designed this case report. The remaining authors (RM, FN, YO, KW, DT, SN, YZ, YK, TY, KM, and TN) contributed to the collection, analysis, and interpretation of the data. TT wrote the draft of the manuscript, and MN performed the critical revision of the manuscript. TN gave the final approval of the version to be published. MN took overall responsibility and guaranteed the scientific integrity of the manuscript. All authors read and approved the final manuscript.

Funding
No funding was received in support of this work.

Availability of data and materials
All data generated or analyzed during this study are included in this published article.

Ethics approval and consent to participate
Informed consent was obtained from the patient.

Consent for publication
Informed consent to publish was obtained from the patient.

Competing interests
The authors declare that they have no competing interests.

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Received: 5 June 2019 Accepted: 1 October 2019

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