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Ectopic Hepatocellular Carcinoma in an Unusual Retroperitoneal Location: Case Report and Review of Published Evidence

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ABSTRACT

Hepatocellular carcinoma developing in aberrant locations (EHCC) exhibits a morphologic and immunohistochemical profile similar to that of classic intrahepatic hepatocellular carcinoma (HCC). Yet, it springs from ectopic liver tissue (EL)—hepatic parenchyma anatomically detached from the main hepatic mass. Due to its scarcity, EHCC is difficult to identify preoperatively. Consequently, its distribution across populations, optimal management, and long-term outcomes remain poorly mapped. In this contribution, we detail the clinical course of a 52-year-old man found to have unresectable EHCC, who received transarterial chemoembolization (TACE) and ultimately died 13 months post-intervention, supplemented by a review of published reports that consolidates the epidemiological picture and scrutinizes treatment pathways and survival endpoints pooled from documented case data.

Keywords: Ectopic liver, Ectopic hepatocellular carcinoma, Hepatocellular carcinoma, Metastasis, Transarterial chemoembolization, Case report

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Introduction

Hepatic tissue displaced beyond the orthotopic site that retains ductal continuity with the biliary apparatus is termed the accessory liver. In contrast, parenchyma fully sequestered from the biliary conduits is known as ectopic liver (EL) [1, 2]. Recognized anatomic niches for EL encompass the gallbladder, spleen, pancreas, adrenal gland, Arantius ligament, diaphragm, thorax, retroperitoneum, and omentum [3]. Compared with the orthotopic liver, EL carries an amplified malignant potential stemming from its paucity of normal vascular inflow and defective bile clearance [2]; it has been hypothesized that EHCC tumorigenesis is linked to faulty biliary egress and/or restricted perfusion of the EL [4]. Meanwhile, classic drivers of HCC originating in the native liver—including cirrhosis, hepatitis virus infection, or other well-established triggers—hold less bearing on EHCC [2, 5]. The precise molecular underpinnings, however, remain elusive and require dedicated exploration.

Even though HCC stands as the most widespread primary hepatic cancer and occupies the fifth slot among cancer-attributable deaths globally [6], its manifestation within EL—autonomous hepatic units completely disjoined topographically from the maternal organ—represents an extraordinary rarity among clinicopathologic diagnoses [3]. The literature holds merely a scattering of such accounts. We present here a case of non-operable EHCC treated with transarterial chemoembolization (TACE) and perform a structured survey of published cases to map epidemiological trends and dissect therapeutic regimens alongside prognostic trajectories derived from previously reported cases.

Case presentation

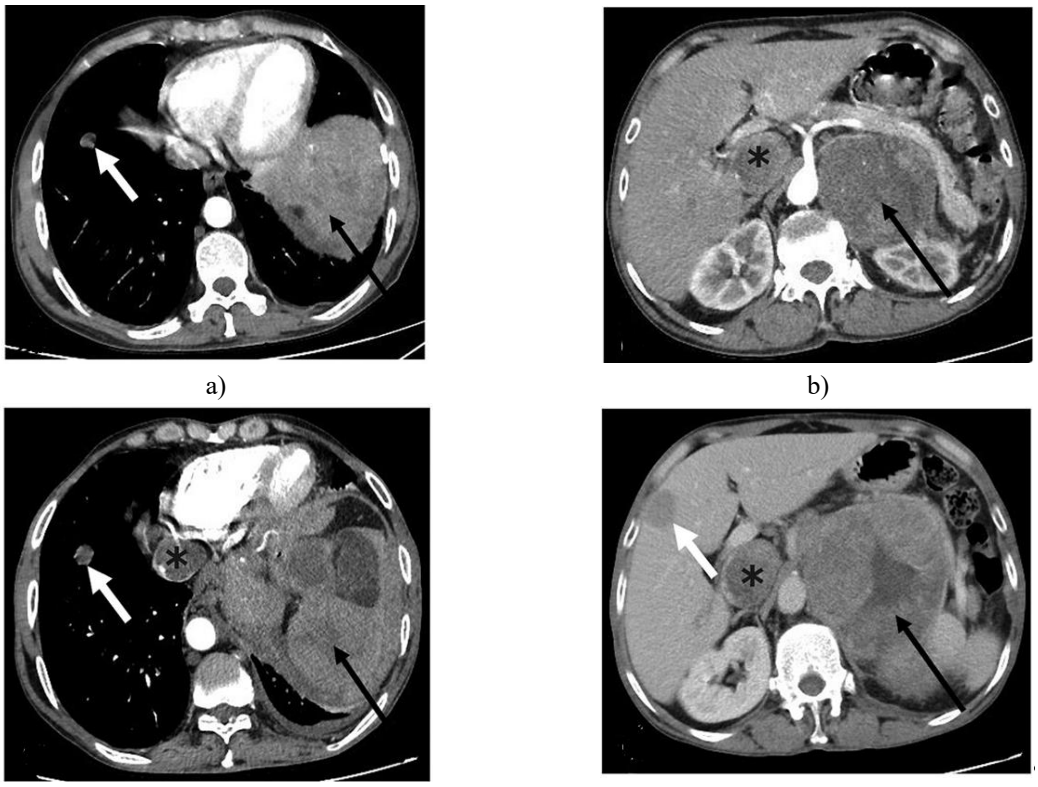
A 52-year-old male originally sought care three months earlier at Quanzhou People's Hospital of Fujian Province (China) with a chief complaint of abdominal discomfort. Abdominal contrast-enhanced computed tomography (CECT) imaging led to a radiological impression of an intraperitoneal and retroperitoneal mass consistent with malignancy. Upon learning the seriousness of the finding, the patient formed the conviction that pursuing medical care would be pointless and left the hospital on his own accord, without continuing treatment. Through the three-month window following discharge, his symptoms escalated to frequent, severe discomfort localizing to the left upper quadrant of the abdomen, motivating his current presentation to our facility for pain control. He acknowledges nightly intake of 250 mL of 53° spirits stretching back roughly 30 years and daily use of 40 cigarettes over approximately 20 years. The family history was non-contributory. An HBV infection came to light during the admission workup, leading to the start of Entecavir capsules (0.5 mg once daily by mouth). Bedside evaluation showed unremarkable vital parameters aside from focal tenderness and rebound discomfort elicited in the left upper abdominal region. Laboratory panels returned: HBV surface antigen (HBsAg), 159.860 (reference: < 0.05) IU/mL; HBV surface antibody (HBsAb), 0.350 (reference: < 10) mIU/mL; HBVe antigen (HBeAg), 0.362 (reference: < 1) COI; HBV e antibody (HBeAb), 0.01 (reference: < 1) COI; HBV core antibody (HBcAb), 10.06 (reference: < 1) COI; HBV DNA, 6.512×10^2 (reference: $< 3 \times 10$) IU/mL; hepatitis C virus antibody (HCV Ab), negative. Tumor-associated circulating markers were documented as: alpha-fetoprotein (AFP), 39.2 (reference: < 9) ng/mL; ferritin, 679 (reference: 23.9–336.2) μg/L; carbohydrate antigen 19–9 (CA19–9), carbohydrate antigen 72–4 (CA72–4), and carcinoembryonic antigen (CEA), all within the normal range **(Table 1).**

Table 1. Laboratory tests and tumor markers.

Laboratory tests			Tumor marker		
Variables (Unit)	**Reference range**	**Result**	**Variables (Unit)**	**Reference range**	**Result**
HBsAg (IU/mL)	< 0.05	159.860	AFP (ng/mL)	< 9	39.2
HBsAb(mIU/mL)	< 10	0.350	Ferritin (μg/L)	23.9~336.2	679
HBeAg (COI)	< 1	0.362	CA19–9 (U/mL)	< 25	16
HBeAb (COI)	< 1	0.01	CA72–4 (U/mL)	< 6.9	0.8
HBcAb (COI)	< 1	10.06	CEA (μg/L)	< 5	3.4
HBV DNA(IU/mL)	$< 3\times10^1$	6.512×10^2			
HCV Ab	–	negative			

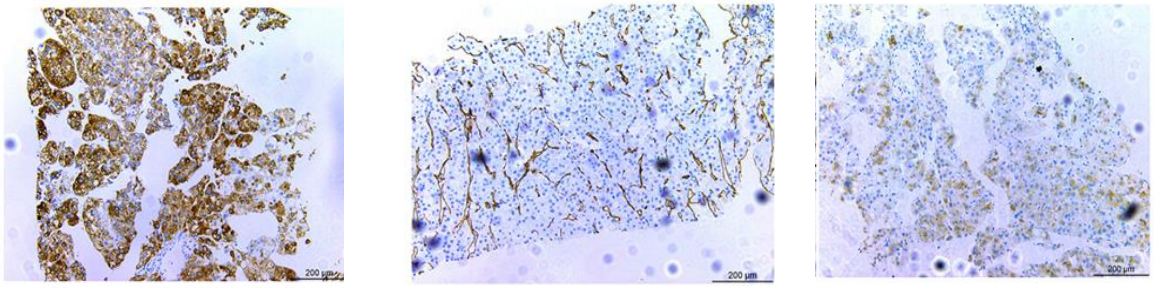
The CECT scan of the abdomen, taken on October 19, 2021, at Quanzhou People's Hospital, displayed a soft tissue mass of irregular contour situated in the left sub-diaphragmatic space **(Figure 1a)**, (black arrows) and extending into the retroperitoneum **(Figure 1b)**, (black arrows). The volume of the sub-diaphragmatic component was recorded at $8.5 \times 9.3 \times 7.8$ cm³, while the retroperitoneal portion measured $8.3 \times 7.5 \times 12.1$ cm³, with both showing inconsistent uptake of contrast agent. Notable widening of the inferior vena cava (IVC) was present, and within it, a tumor thrombus demonstrated patchy enhancement **(Figure 1b)**, (asterisks). The mother's liver, notably, revealed no evident pathology. A subsequent examination at our facility (Affiliated Hospital of Zunyi Medical University, Guizhou Province, China) on January 10, 2022, identified expansion of the sub-diaphragmatic **(Figure 1c)**, (black arrows) and retroperitoneal **(Figure 1d)**, black arrows) lesions to dimensions of $11.2 \times 11.5 \times 9.8$ cm³ and $11.3 \times 9.5 \times 14.5$ cm³, respectively. Within the IVC, numerous tumor thrombi with heterogeneous enhancement were now apparent **(Figures 1c and 1d)**, (asterisks). Concurrent findings included multiple, round, hyperdense nodules in both lung fields that enhanced slightly **(Figures 1a and 1c)**, (white arrows) and a host of hypodense hepatic foci displaying faint, rim-like contrast uptake **(Figure 1d)**, (white arrows), though without classic HCC imaging features. Nodal enlargement was observed in the mediastinum, bilateral lung hila, and retroperitoneum, with node diameters ranging from 0.5 to 1.5 cm and mild enhancement. A comparison with the external study from three months prior made the aggressive progression clear: the primary left subdiaphragmatic and retroperitoneal masses were distinctly larger, lung metastases were more numerous and enlarged, and intrahepatic metastases were a new finding. The radiological conclusion was a left abdominopelvic and retroperitoneal malignancy complicated by extensive IVC tumor thrombus burden and metastatic spread to the liver and pulmonary tissues. On January 13, 2022, a CT-guided percutaneous core biopsy of the abdominal mass was performed to clarify its histogenesis. The pathologic review, finalized three days later, confirmed the tissue as HCC (Class II). The IHC panel exhibited the following profile: CK (+), Hep Par-1 (+), Arginase-1(-),

AFP (-), GPC-3(+), CK7 (-), CR (-), a-inhibin (-), CgA (-), Syn (±), and CD34 delineating hepatic-type sinusoidal vascular channels **(Figure 2)**. The tumor's cellular positivity for Hep Par-1 **(Figure 2a),** the sinusoidal vascular pattern highlighted by CD34 **(Figure 2b),** and the expression of GPC-3 **(Figure 2c)** each pointed toward a hepatic tissue origin. This was despite the liver appearing entirely healthy on the CECT from three months earlier, and the later-emerging, vaguely enhancing intrahepatic lesions not satisfying radiological criteria for primary HCC. Since no physical continuity between the mother's liver and the masses was discernible on CT imaging, the fusion of radiographic and pathologic evidence solidified a final diagnosis of EHCC of the left sub-diaphragmatic and retroperitoneal spaces, with IVC tumor thrombosis and hepatic and pulmonary metastases.



a) b) c) d)

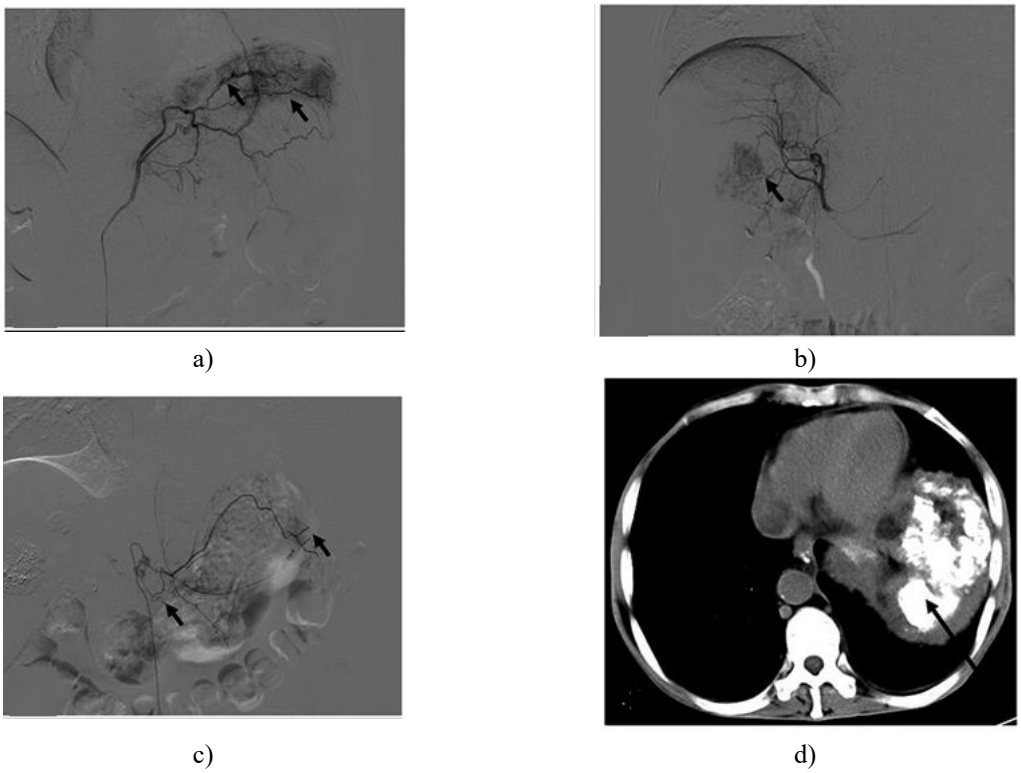
Figure 1. CECT images: (a–d): CECT of the abdomen shows an irregular soft tissue mass in the left sub-diaphragmatic area (a, c: black arrows) and retroperitoneum (b, d: black arrows) with multiple tumor thrombus formations in the IVC (b, c, d: asterisks) and multiple metastases in the lungs (a, c: white arrows) and liver (d: white arrows). A and B are 3 months before the comparison, and C and D are 3 months after it. Intrahepatic metastases were newly added, and other masses were significantly enlarged. Abbreviations: CECT = contrast-enhanced computed tomography; IVC = inferior vena cava.



a) b) c)

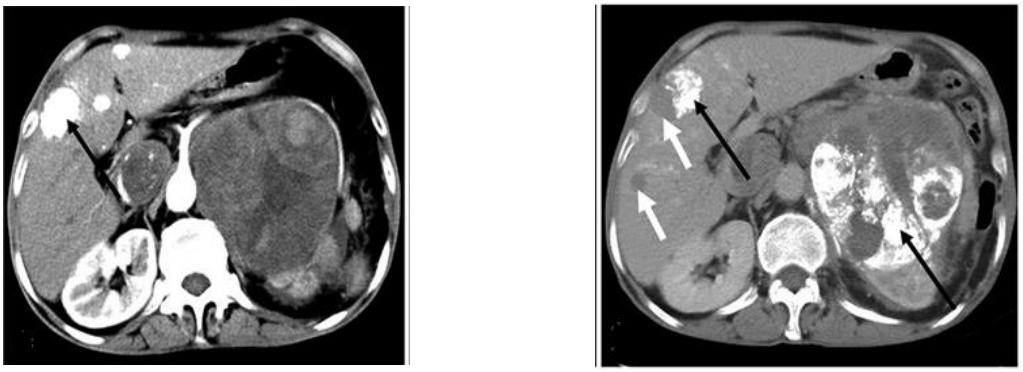
Figure 2. IHC: (a–c): IHC shows that the tumor cells were positive for hep per-1 (a), CD34 demonstrates hepatic sinusoidal vascularization (b), and GPC-3 (c). Abbreviation: IHC = immunohistochemistry.

The therapeutic course commenced with TACE on January 23, 2022. During the procedure, DSA mapping of the abdominal arteries revealed a hypertrophied, serpentine left inferior phrenic artery feeding the left subdiaphragmatic tumors, which produced a distinct stain **(Figure 3a),** (arrows). The retroperitoneal component was supplied by similarly hypertrophied and tortuous branches arising from the left adrenal and left renal arteries **(Figure 3c)**, (arrows). The proper hepatic artery branches, which were thickened and coiled, vascularized the intrahepatic metastases in a pattern typical of such lesions **(Figure 3b)**, (arrows). Given the extensive nature of the left sub-diaphragmatic, retroperitoneal, and intrahepatic disease, TACE was clinically indicated. Embolizing the intraperitoneal and retroperitoneal sectors in a single session, however, was considered high-risk due to total tumor volume; thus, the decision was made to select Shanghai Xudong Haipu Pharmaceutical Co., LTD. China) + 30 mg epirubicin suspension (Shandong New Times Pharmaceutical Co., LTD., China) to embolize the left sub-diaphragmatic mass and the intrahepatic metastases specifically. A post-procedural regimen of glycyrrhizic acid monoamine S, rabeprazole for gastric protection, and tropisetron hydrochloride injection for nausea was given. The patient's abdominal pain had improved by one week, allowing discharge. Follow-up abdominal CECT on February 18, 2022, revealed that the left sub-diaphragmatic **(Figure 3d)**, (black arrows) and intrahepatic **(Figure 3e)**, (black arrows) target areas showed dense radiopaque material but no signs of pathological enhancement. The unaddressed retroperitoneal mass was stable compared to three months prior. Hospitalization for a second TACE episode targeting the retroperitoneal disease occurred on February 20, 2022. A re-staging CT on March 30, 2022, verified dense, stable opacification of the left sub-diaphragmatic, retroperitoneal, and intrahepatic treated territories. The emergence of new intrahepatic metastatic nodules (**Figure 3f)**, (white arrows) was documented, alongside a minimal increase in the retroperitoneal tumor bulk. Overall, the two-month post-TACE radiological assessment indicated no marked disease progression. The patient was then discharged in stable condition with less abdominal pain and without any post-TACE complications such as hepatic failure, renal impairment, or myelosuppression. Death from the disease occurred at the 13-month follow-up mark. The entire case timeline is illustrated in **Figure 4.**



a) b)

c) d)



e) f)

Figure 3. DSA and CECT images: (a–c) DSA of abdominal vessels shows the left inferior phrenic artery (a: black arrows), branches of the left adrenal artery (c: black arrows), and the proper hepatic artery (b: black arrows) were thickened, seem to be tortuous, and are involved in the blood supply of the left sub-diaphragmatic, retroperitoneal, and intrahepatic tumors, respectively. d, e, f: CECT shows high-density shadow deposition in the left sub-diaphragmatic (d: black arrows), retroperitoneal (fig. f black arrows), and intrahepatic (fig. e black arrows) lesions after TACE and increased and enlarged intrahepatic metastases (f white arrows). Abbreviations: CECT = contrast-enhanced computed tomography; DSA = digital subtraction angiography; TACE = transarterial chemoembolization.

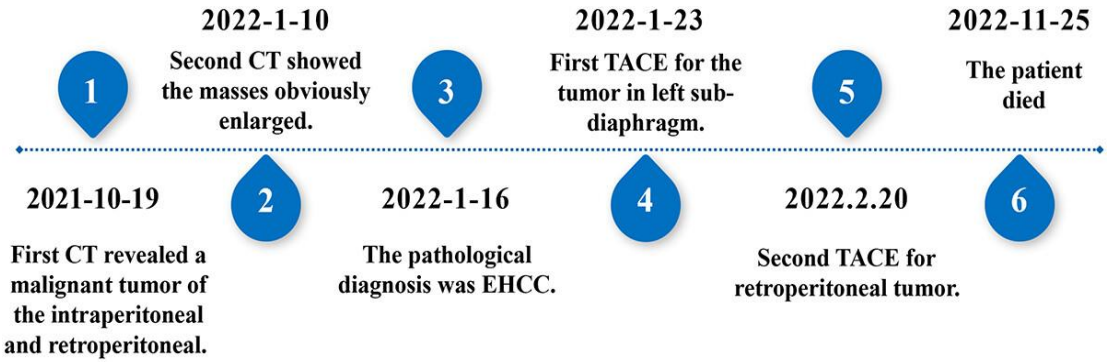


Figure 4. Timeline.

In this presentation, the sole factor linking the patient to hepatic malignancy was HBV; no additional clinical signs, radiographic stigmata, or serologic derangements indicative of orthotopic HCC were identified. One also notes that earlier publications on EHCC have predominantly depicted small-volume tumor deposits, whereas our subject harbored a remarkably large disease burden. An added layer of complexity stemmed from tumor involvement of both the peritoneal cavity and the retroperitoneal compartment, rendering accurate preoperative recognition exceptionally problematic. Despite these diagnostic hurdles, the current account constitutes, to the best of our understanding, the inaugural report of EHCC simultaneously invading the abdominal cavity and retroperitoneal space.

Turning to the literature synthesis, a systematic search of PubMed for English-language papers published from 2015 through March 31, 2022, using the search term “Ectopic hepatocellular carcinoma,” retrieved 616 candidate records. The filtering cascade is mapped out in **Figure 5.** Ultimately, 14 cases satisfied the eligibility benchmarks and were taken forward for aggregation. Variables harvested from each report encompassed lead author, year of publication, age, sex, anatomic site, predisposing factors, AFP values, treatment rendered, and survival status at last contact **(Table 2).**

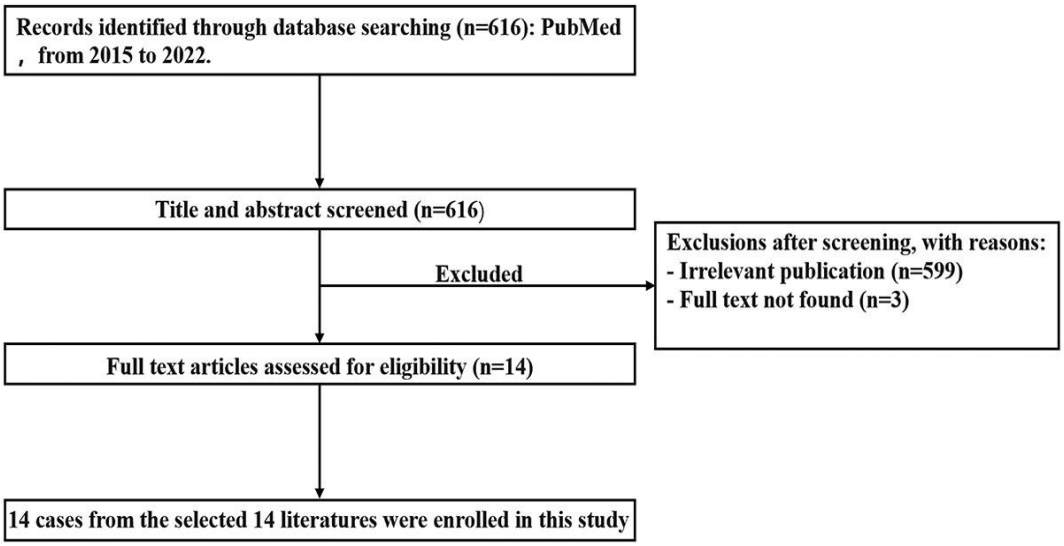


Figure 5. Flowchart of the literature screening process for EHCC. Abbreviations: EHCC = ectopic hepatocellular carcinoma.

Table 2. Summary of reported cases of EHCC.

Reference	Age (years)	Sex	Location	Risk factors	AFP (ng/mL)	Diagnostic methods	Treatment	Outcome
Vining *et al.* [2]	67	M	Pancreas	HBV(-), HCV(-)	Normal	Surgical resection	Resection	ND
Adachi *et al.* [3]	81	F	Retroperitoneal	HCV (+)	30.1	Surgical resection	Resection	Alive at 8 months
Ko *et al.* [5]	73	M	Peritoneum	HBV(-), HCV(-)	>1000	Laparoscopic resection	Sorafenib	Dead at 1 year
Aarås *et al.* [7]	64	F	Peritoneum	HBV(+), HCV(-)	200	Laparoscopic resection	Resection	Alive at 4 years
Cui *et al.* [8]	63	M	Intrathoracic Intraperitoneal	HBV(+)	24793	Surgical resection	Resection Sorafenib	Alive at 13 months
Cheng *et al.* [9]	54	M	Bile Duct	HBV (+)	3724.75	Surgical resection	Resection	Alive at 3 months
Rorris *et al.* [10]	53	M	Adrenal	HCV (+)	Normal	Surgical resection	Resection	ND
Li *et al.* [11]	44	F	Pancreas	HBV(-), HCV(-)	>1200	Surgical resection	Resection	Alive at 21 months
Lee *et al.* [12]	65	M	Peritoneum	HBV(-), HCV(-)	ND	Surgical resection	Resection	Alive at 17 months
George *et al.* [13]	69	M	Choledochal	HBV (-)	2.9	Biopsy	Resection	ND
Jin *et al.* [14]	56	M	Peritoneum	HBV(-), HCV(-)	8.03	Surgical resection	Resection	Dead at 22 months
Braun *et al.* [15]	77	M	Pancreas	HBV(-), HCV(-)	Normal	Surgical resection	Resection	Alive at 2 years
Wei *et al.* [16]	71	M	Adrenal	HBV(-), HCV(-)	1.87	Surgical resection	Resection Lenvatinib	Alive at 10 months
Allencherril *et al.* [17]	82	M	Chest Wall	HBV(-), HCV(-)	ND	Biopsy	No further treatment	Dead at 2 weeks
Present case	52	M	Peritoneum Retroperitoneal	HBV(+)	39.2	Biopsy	TACE	Dead at 13 months

Abbreviations: EHCC = Ectopic hepatocellular carcinoma; AFP = Alpha-fetoprotein; F = Female; M = Male; HBV = Hepatitis B virus; HCV = Hepatitis C virus; TACE = Transarterial chemoembolization.

The assembled series comprised 11 men and 3 women, yielding a mean presentation age of 65.6 years (range, 44-82 years). The predilection for older age brackets mirrors what is typically observed with HCC native to the liver. Nonetheless, a firmer conclusion regarding any sex-based disparity in EHCC occurrence awaits a more robust

body of case material. Tumor locations across these reports were distributed among the peritoneum, retroperitoneum, intraperitoneal cavity, adrenal gland, pancreas, bile duct, thoracic cavity, and chest wall, matching the distribution noted in prior literature [3]. Of the 14 individuals, a background of viral hepatitis was documented in only 5: 3 carried HBV [7-9] and 2 were afflicted with HCV [3, 10]. This distribution reinforces the narrative that classic hepatocarcinogenic drivers—such as HBV, HCV, heavy alcohol intake, and cirrhosis—exhibit markedly diminished relevance in EHCC pathogenesis [5]. Laboratory evidence of AFP elevation was noteworthy in merely 4 subjects among the 14 [5, 8, 9, 11]. Conventionally adopted as both a diagnostic and prognostic serum marker for HCC, AFP concentrations exceed normal thresholds in an estimated 60% of primary HCC diagnoses [18]. Li *et al*. [11] have argued that AFP should be considered an equally crucial biomarker in the context of EHCC. Yet the present collation suggests that the prevalence of serum AFP abnormalities may be lower than the rates documented for primary HCC. This phenomenon only deepens the preoperative diagnostic challenge posed by EHCC and underscores the need for corroboration through additional cases.

A survey of global treatment practices reveals that therapeutic strategies for EHCC coalesce around surgical extirpation and molecularly targeted systemic agents, namely Sorafenib and Lenvatinib [2, 3, 5, 7-17]. Ten of the 14 subjects underwent surgical resection as standalone therapy [2, 3, 7, 9-17]; of the remaining four, adjuvant Sorafenib was prescribed post-resection in one instance [8], one patient received surgery followed subsequently by Lenvatinib [16], and one was managed with Sorafenib monotherapy [5]. A single case had no further oncologic intervention whatsoever [17]. Standardized management algorithms and consensus recommendations remain absent for EHCC, largely owing to the sheer scarcity of data. Vining *et al*. [2] advanced the principle that operative excision offers definitive treatment whenever the EL-derived HCC is technically resectable. On this premise, many patients lacking distant dissemination have undergone surgical clearance and achieved favorable long-term trajectories. Hitherto, no case of EHCC presenting with widely metastatic deposits has been reported, leaving a knowledge gap regarding therapy for such advanced scenarios. The generally poor outlook for EHCC underscores the urgency of crafting a rational therapeutic approach. Lee *et al*. [12] observed that most managing clinicians extrapolate the treatment paradigms established for native-liver HCC. Moreover, Chen *et al*. [6] underscored that early-stage HCC patients are eligible for potentially curative modalities, including transplantation, hepatic resection, or local ablation.

In contrast, those classified as intermediate-stage are directed toward TACE as the preferred first-line intervention, in alignment with prevailing European and American practice frameworks. Guided by these considerations, we opted to deploy TACE in our case. Over the two TACE cycles, the patient's clinical trajectory, which had been marked by rapid worsening in the antecedent three months, slowed perceptibly. The overall tumor status remained largely contained, although intrahepatic metastatic foci did appear and expand; meanwhile, the left sub-diaphragmatic and retroperitoneal masses did not exhibit meaningful progression. To our knowledge, this account marks the inaugural documentation of TACE administered for EHCC. Consequently, the evidence base does not yet support a confident endorsement of TACE as the principal therapy for intermediate-stage EHCC, and rigorous evaluation of subsequent cases will be required.

Conclusion

As this synthesis reveals, the phenomenon of EHCC seeding multiple metastases in the mother's liver and lungs is extremely rare. The great majority of individuals diagnosed with EHCC lack underlying cirrhosis or any of the widely recognized causative triggers linked to conventional HCC. Moreover, establishing a confident diagnosis before operative intervention remains a formidable undertaking, constrained by the unpredictable anatomic location of the tumor and the entity's sheer infrequency; definitive diagnosis thus remains contingent upon histopathologic examination. To date, no standardized protocols or consensus-driven treatment guidelines have emerged for the management of EHCC. Where resectability is confirmed, prompt and complete surgical excision is warranted. In contrast, efficacious therapeutic options for unresectable EHCC remain undefined. Consequently, gathering and analyzing additional cases is essential to delineate the role, if any, that TACE can play in the treatment landscape for unresectable EHCC.

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Conflict of Interest: None

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