Journal of Hepatocellular Carcinoma

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CASE REPORT

Gastropleural Fistula Following Combined TACE, Immunotherapy, and Bevacizumab in HCC: A Case Report

Xin Xu, Jingjing Li, Lanyue Pan, Haijing Yu*, Jiaquan Huang*

Department of Infectious Diseases, Tongji Hospital, Tongji Medical College and State Key Laboratory for Diagnosis and Treatment of Severe Zoonotic Infectious Diseases, Huazhong University of Science and Technology, Wuhan, Hubei, 430030, People's Republic of China

*These authors contributed equally to this work

Correspondence: Jiaquan Huang; Haijing Yu, Department of Infectious Diseases, Tongji Hospital, Tongji Medical College and State Key Laboratory for Diagnosis and Treatment of Severe Zoonotic Infectious Diseases, Huazhong University of Science and Technology, No. 1095 Jie Fang Avenue, Qiaokou, Wuhan, Hubei, 430030, People's Republic of China, Email huangjiaquan21@aliyun.com; 463104640@qq.com

Abstract: Gastropleural fistula (GPF) is an extremely rare complication after treatment for liver cancer. We report a case of a 54-year-old man with hepatitis B virus (HBV)-related liver cancer who developed a GPF after multiple sessions of transarterial chemoembolization (TACE) combined with immunotherapy and targeted therapy. During the third treatment, because of arterial remodeling and changes in tumor vascular supply, the embolization route was changed to the left inferior phrenic artery. After the procedure, the patient presented with abdominal pain, chest pain, and fever. Metagenomic next-generation sequencing (mNGS) of the pleural effusion identified Porphyromonas endodontalis, and Pneumocystis jirovecii was also detected in the sputum. Upper gastrointestinal endoscopy and water-soluble contrast radiography confirmed a gastric fundus perforation with a fistulous communication to the pleural cavity. After multidisciplinary evaluation, the patient underwent laparoscopic fistula repair and had a favorable postoperative recovery. This case highlights that, while the combination of TACE, immunotherapy, and targeted agents may provide synergistic antitumor benefits, it also carries a potential risk of serious gastric complications.

Keywords: liver cancer, TACE, sintilimab, bevacizumab, gastropleural fistula, adverse event, case report

Introduction

Liver cancer is the sixth most commonly diagnosed malignancy worldwide and the third leading cause of cancer-related death.[1] Because many of patients are diagnosed at an intermediate or advanced stage and are not candidates for curative surgery, transarterial chemoembolization (TACE) is commonly employed as a locoregional, non-surgical treatment modality.[2,3] In recent years, the combined use of immune checkpoint inhibitors (ICIs) and antiangiogenic drugs has offered new therapeutic options for patients with liver cancer.

Clinical trials such as IMbrave150 and ORIENT-32 have demonstrated that combining PD-1/PD-L1 inhibitors with bevacizumab can significantly improve overall survival (OS) and progression-free survival (PFS),[4–6] leading to global approval of this systemic regimen as a first-line treatment for advanced liver cancer.[7] The CHANCE2201 study further showed that TACE effectively reduces intrahepatic tumor burden and helps establish a more favorable immune microenvironment for systemic therapy, suggesting that combining TACE with systemic treatment may prolong OS through synergistic mechanisms.[8] However, while this approach enhances antitumor efficacy, it may also increase gastrointestinal risk and lead to severe adverse events.

Here, we report a patient with HBV-related liver cancer who developed a Gastropleural fistula (GPF), complicated by opportunistic infections, after receiving multiple sessions of TACE in combination with sintilimab and a bevacizumab

Received: 15 August 2025
Accepted: 26 November 2025
Published: 3 December 2025

biosimilar. In this report, we explore the possible mechanisms underlying GPF and share lessons from this rare but serious complication.

Case Presentation

In February 2025, a 54-year-old man was admitted with a 20-day history of abdominal pain. He had a history of chronic hepatitis B virus infection for over two decades and was undergoing antiviral therapy with tenofovir alafenamide fumarate and entecavir. His medical history also included a family history of liver cancer. Approximately two months earlier, a routine outpatient abdominal CT scan revealed liver cirrhosis and multiple hepatic lesions, the largest measuring 48.1×59.2 mm, which raised concern for malignancy. Laboratory tests revealed elevated tumor markers, including alpha-fetoprotein (AFP) of 57.41 ng/mL and protein induced by vitamin K absence or antagonist-II (PIVKA-II) of 4045.44 mAU/mL, supporting a clinical diagnosis of liver cancer. Given his preserved liver function (Child-Pugh class A), Eastern Cooperative Oncology Group (ECOG) performance status of 0, and Barcelona Clinic Liver Cancer (BCLC) stage B, the patient was considered suitable for TACE combined with systemic therapy. Following multidisciplinary consultation, the patient received two cycles of TACE combined with sintilimab and IBI305—a bevacizumab biosimilar approved for advanced liver cancer—in November and December of 2024. During each TACE procedure, a microcatheter was selectively placed into the tumor-feeding branches of the left and right hepatic arteries. The procedure involved intra-arterial infusion of 10 mL of lipiodol and 50 mg of lobaplatin, followed by embolization with gelatin sponge particles. Systemic therapy consisted of intravenous sintilimab (200 mg) and IBI305 (600 mg).

At the time of his third hospital admission, follow-up contrast-enhanced liver MRI and tumor marker levels demonstrated improvement compared with previous evaluations. Angiography during the third TACE procedure revealed a narrowed left hepatic artery and a prominent collateral supply from the left inferior phrenic artery, accompanied by abnormal tumor blush. A microcatheter was selectively advanced into the tumor-feeding branches of the left inferior phrenic artery and right hepatic artery, followed by intra-arterial infusion of 10 mL of lipiodol, 50 mg of lobaplatin, and an appropriate amount of gelatin sponge particles. Post-embolization angiography confirmed satisfactory lipiodol retention within the tumor. The immunotherapy and targeted therapy regimen remained unchanged. The patient recovered well and was discharged in stable condition after completion of the third treatment session.

Twenty days before admission, the patient developed intermittent upper abdominal pain. On admission, his temperature was 37.7°C, oxygen saturation was 98%, and lung auscultation revealed no remarkable abnormalities. Abdominal examination revealed no significant tenderness nor rebound pain. Chest computed tomography (CT), compared with imaging from his previous admission (Figure 1A), revealed new left lung atelectasis and a newly developed loculated pleural effusion on the left (Figure 1B). Empirical antimicrobial therapy with meropenem was initiated. On hospital day 3, the patient developed a high-grade fever (39.3°C), accompanied by chest pain and dyspnea. To relieve respiratory distress, ultrasound-guided left-sided thoracic drainage was performed, yielding approximately 100 mL of slightly reddish fluid. Cytologic analysis of the pleural fluid showed a background of red blood cells and necrotic debris. Numerous neutrophils, scattered mesothelial cells, and occasional macrophages were observed. No malignant or atypical cells were identified. Biochemical analysis of the pleural fluid revealed the following values: albumin, 20.9 g/L; lactate dehydrogenase (LDH), 1569 U/L; and adenosine deaminase (ADA), 34 IU/L. Metagenomic next-generation sequencing (mNGS) of the pleural fluid identified *Porphyromonas endodontalis* with a relative abundance of 74.1%, suggesting it as a potential pathogen. Subsequent sputum analysis identified *Pneumocystis jirovecii*, prompting escalation of antimicrobial therapy to include sulfonamides in combination with caspofungin.

After adjustment of the antimicrobial regimen, the patient's body temperature remained between 36.4°C and 37.8°C, accompanied by partial relief of abdominal and chest pain. Repeat chest CT revealed an encapsulated air-fluid collection in the left pleural space, with decreased effusion and markedly increased intrathoracic air compared with prior imaging (Figure 1C and D). Radiology consultation raised concern for a left-sided diaphragmatic hernia with partial herniation of bowel loops into the thoracic cavity as the likely source of the increased air. A subsequent abdominal CT raised suspicion of a GPF, which was later confirmed by upper gastrointestinal contrast examination with a water-soluble agent (Figure 2A). Esophagogastroduodenoscopy revealed a large ulcerative lesion on the anterior wall at the junction of the gastric body and fundus, with a visible fistulous opening at its base (Figure 3A and B). Biopsies obtained from the ulcer

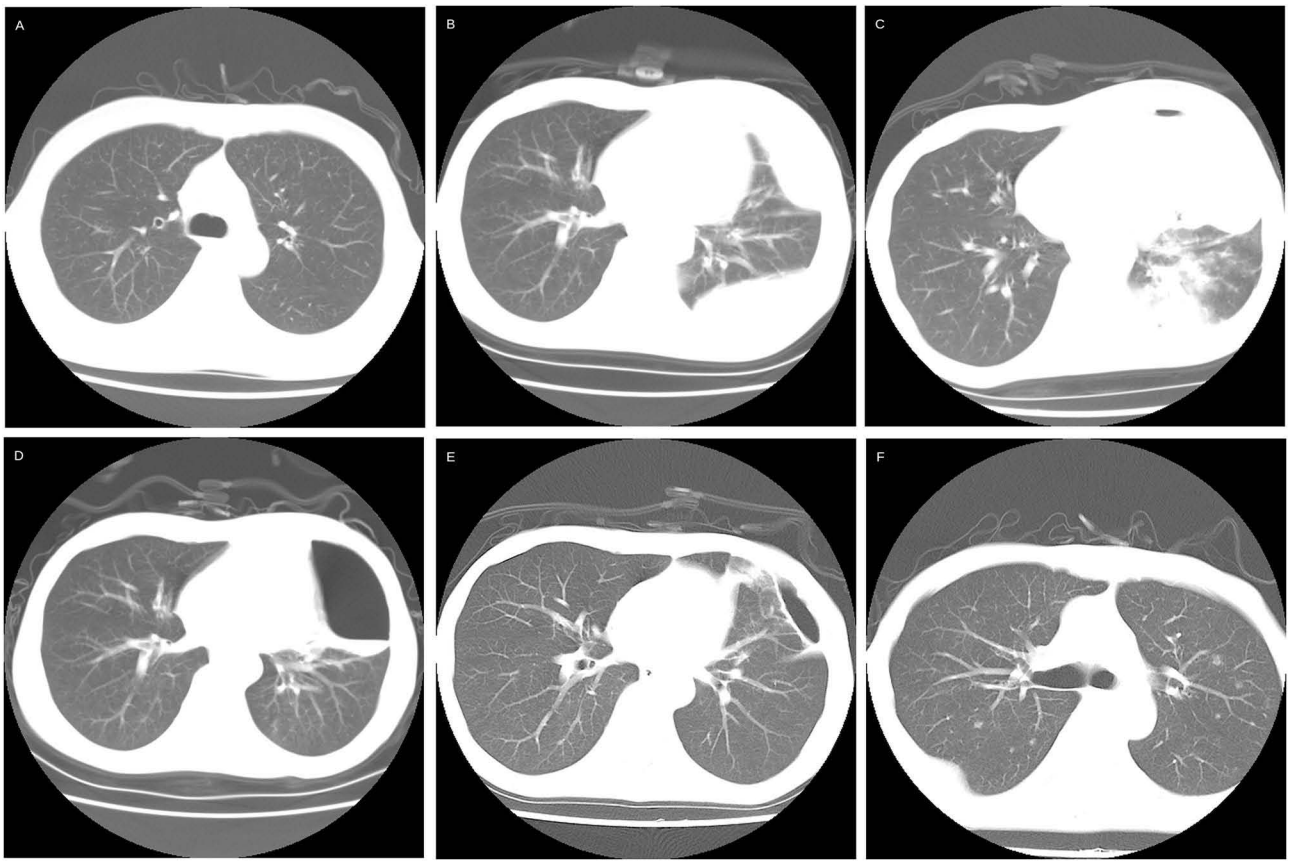

Figure 1 Chest CT images. (**A**) Normal lungs during the third hospitalization. (**B**) Development of pleural effusion. (**C** and **D**) Progressive changes during disease progression. (**E**) Improvement with conservative treatment. (**F**) Postoperative follow-up showing near-complete restoration of normal lung appearance.

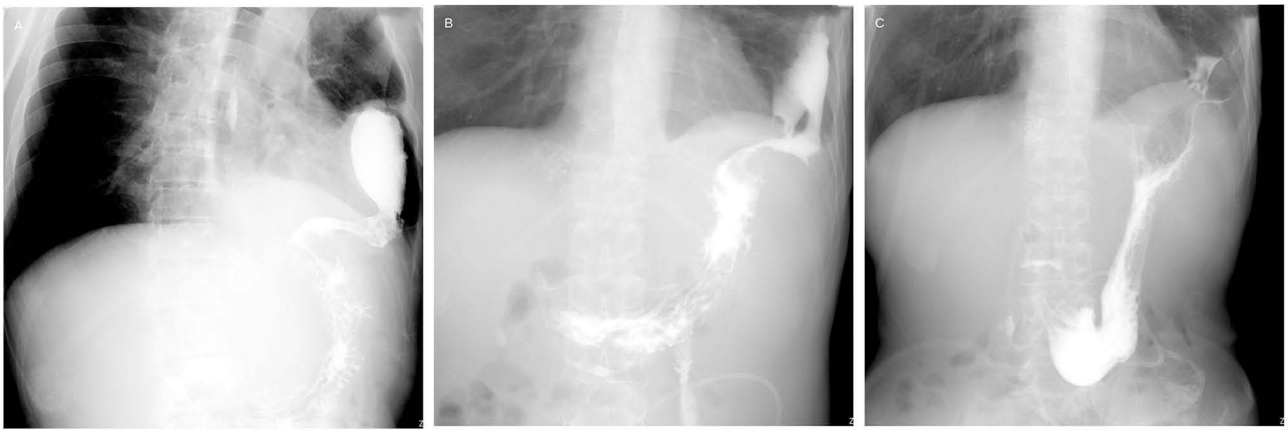

Figure 2 Water-soluble contrast images. (**A**) Initial study confirming a gastropleural fistula. (**B** and **C**) Progressive improvement following conservative treatment.

margin showed features consistent with ulceration. A jejunal feeding tube was placed endoscopically to divert enteral contents and prevent pleural contamination with secondary infection. Given the limited response to conservative treatment (Figures 1E, 2B, C and 3C), a multidisciplinary team consultation was initiated. The patient underwent laparoscopic resection of the gastric lesion with concurrent closed thoracic drainage under joint care of thoracic and gastrointestinal surgeons, successfully achieving fistula closure. The chest tube was removed on postoperative day 9, and the patient was discharged in stable condition. On follow-up chest CT two months after surgery, the previously noted

 https://doi.org/10.2147/JHC.S560287

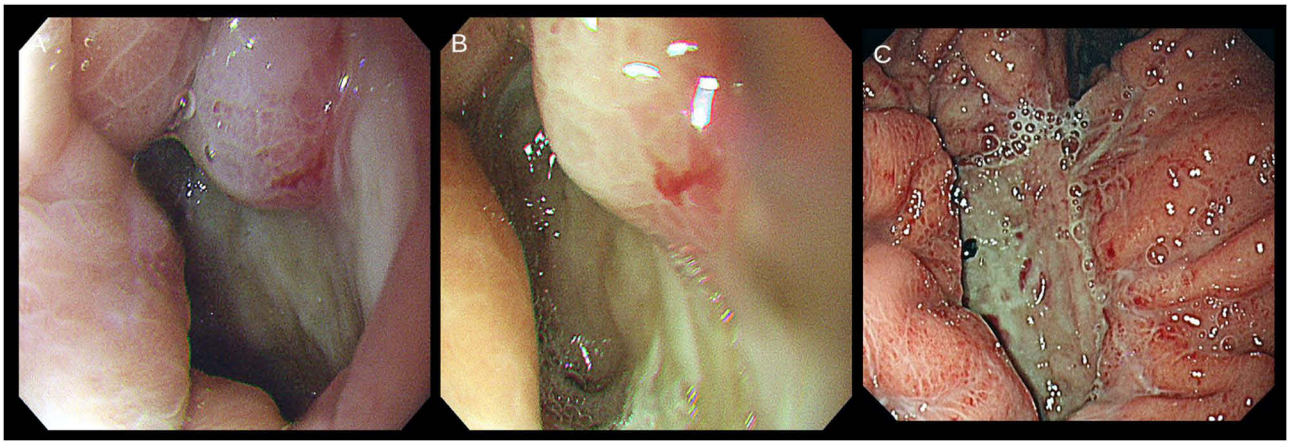

Figure 3 Gastroscopic images. (**A** and **B**) Fistula orifice visible in the gastric fundus. (**C**) Improvement after conservative treatment.

pleural air–fluid cavity had essentially returned to normal, with complete resolution of effusion and restoration of left lung expansion (Figure 1F).

Discussion

Driven by global population growth and aging, the incidence and mortality of liver cancer are projected to increase by over 55% by 2040.[9] In response to the escalating global burden of liver cancer, therapeutic strategies are evolving to address new clinical challenges. The IMbrave150 trial demonstrated that atezolizumab combined with bevacizumab significantly improved OS and PFS when compared with sorafenib.[4] The ORIENT-32 study further validated the efficacy and safety of sintilimab plus IBI305 in patients from China.[6] Based on these studies, recent international and domestic guidelines have recommended combining ICIs with antiangiogenic drugs as first-line therapy for intermediate to advanced liver cancer.[3,7,10] Building on this foundation, TACE combined with immunotherapy and targeted therapy has been increasingly investigated as an integrated strategy in patients with unresectable liver cancer. Clinical studies have shown that this approach can improve OS, PFS, and objective response rate (ORR), compared with either TACE or systemic therapy alone.[8,11] Although generally well tolerated, gastrointestinal bleeding has emerged as a clinically relevant complication. In a multicenter study, gastrointestinal bleeding occurred in 2.9% of patients receiving TACE combined with ICIs and anti-VEGF antibodies or tyrosine kinase inhibitors (TKIs), including grade ≥3 bleeding events in 1.0%.[8] As this combination therapy becomes more widely adopted in clinical practice, striking a balance between therapeutic efficacy and safety has emerged as a key concern.

In this case, a GPF developed after multiple sessions of TACE combined with sintilimab and bevacizumab. GPF is an exceptionally rare yet clinically significant condition characterized by an abnormal communication between the gastric lumen and the pleural cavity. Documented etiologies encompass gastric perforation or peptic ulcer disease, malignant invasion across the diaphragm, traumatic or iatrogenic injury, subphrenic abscess or empyema with diaphragmatic erosion, and treatment-induced ischemic necrosis secondary to chemotherapy or anti-angiogenic therapy such as Bevacizumab. To our knowledge, no prior cases have been reported of GPF occurring in association with TACE combined with PD-1 blockade and Bevacizumab in hepatocellular carcinoma. The development of GPF was not attributable to a single factor but rather to multiple interrelated mechanisms. These included non-target ischemia of the gastric fundus and diaphragm due to altered embolization pathways, mucosal injury worsened by systemic therapy, and subsequent infection. Together, these processes formed a pathogenic cascade—ischemia, mucosal breakdown, and microbial translocation—that ultimately led to gastric contents breaching the diaphragm and establishing an abnormal communication with the pleural cavity.

The change in embolization route during TACE was a critical factor in the development of GPF. Fistulous complications such as hepatogastric fistulas,[12–15] hepatoduodenal fistulas,[16,17] and bronchobiliary fistulas[18,19] have been reported following TACE. In this case, repeated embolization of the left hepatic artery led to vessel narrowing,

while angiography showed prominent abnormal branches arising from the left inferior phrenic artery, prompting a change in the embolization route. The left inferior phrenic artery is a common extrahepatic collateral in liver cancer,[20–22] but it may have anatomical communications with vessels supplying the gastric fundus. If embolic or chemotherapeutic agents inadvertently enter these branches, they can cause gastric mucosal ischemia, leading to ulcer formation and even perforation. Severe complications such as extensive gastric ulcers,[23] gastric perforation,[24] and diaphragmatic perforation[25] have all been reported after TACE. Even when the procedure is performed according to standard technique, non-target injury may still occur due to vascular anatomical variations or retrograde flow of embolic agents.[26] Such non-target embolization is a major contributor to the development of ischemic gastric ulcers,[23,27] thereby setting the stage for eventual perforation.

The synergistic effects of systemic therapy likely contributed to the worsening of mucosal injury. Bevacizumab has been reported to cause gastric perforation[28,29] and, in rare cases, a GPF.[30,31] Bevacizumab may enhance the efficacy of immune checkpoint inhibitors by reversing VEGF-mediated immunosuppression and reshaping the tumor's immune microenvironment.[32,33] However, this combination has also been associated with an increased risk of gastric ulcer perforation.[34] When both TACE-induced non-target injury and systemic therapy–related mucosal compromise simultaneously affect the gastric fundus, their synergistic effect may cause full-thickness damage, ultimately resulting in the formation of a transdiaphragmatic fistula.

Infection likely played a contributory role in both the formation and the persistence of the fistula. Previous studies have suggested that PD-1 inhibitor therapy may increase the risk of developing Pneumocystis jirovecii infection.[35] Porphyromonas endodontalis, an oral anaerobe associated with periodontitis and rarely detected in the pleural cavity,[36,37] was identified through mNGS of the pleural fluid. Its presence suggested a pathological communication between the gastrointestinal tract and the pleural space, providing additional support for the diagnosis of GPF. Persistent infection may impair local tissue repair, promote fistula formation, and delay healing.

Importantly, no published cases to date have reported the development of GPF in liver cancer patients treated with TACE in combination with sintilimab and bevacizumab. This case serves as a cautionary example for risk assessment when using combination therapy. First, after repeated TACE procedures, it is essential to reassess changes in blood supply to the tumor and surrounding structures, including the stomach and diaphragm. Particular caution is warranted when embolization involves nontraditional collateral vessels, such as the left inferior phrenic artery, which may compromise gastric perfusion. Second, prior to initiating systemic therapy, endoscopic evaluation should be performed to assess gastric mucosal integrity, and the combined use of VEGF inhibitors with immunotherapy should be avoided in patients with mucosal injury. Finally, the onset of unexplained chest pain, pleural effusion, or fever during treatment should warrant heightened clinical vigilance for perforation-related complications. Early recognition of fistula formation relies on a combination of thoracoabdominal imaging, endoscopic assessment, and mNGS.

Conclusion

This case underscores the importance of carefully balancing therapeutic efficacy and safety in patients undergoing TACE-based combination therapy for liver cancer, particularly in those with altered vascular anatomy after repeated embolization. Early integration of microbiological testing and imaging evaluation can facilitate timely identification of severe complications such as GPF, enabling prompt intervention and improving treatment safety.

Ethics Approval and Consent to Participate

The case report was approved by the Ethics Committee of Tongji Hospital, Huazhong University of Science and Technology (Approval No. TJ-IRB202505022). Written informed consent for publication of the case details and all accompanying images was obtained from the patient, in accordance with the CARE guidelines.

Acknowledgments

We are grateful to the patient for consenting to share clinical data and images for this case report.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was supported by the National Natural Science Foundation of China (Grant No. 81900542) and the State Key Laboratory for Diagnosis and Treatment of Severe Zoonotic Infectious Diseases (Project No. 2024ZZ10018).

Disclosure

The authors declare that they have no conflicts of interest in this work.

References

1. Bray F, Laversanne M, Sung H, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin*. 2024;74(3):229–263. doi:10.3322/caac.21834
2. Lencioni R, De baere T, Soulen MC, Rilling WS, Geschwind JF. Lipiodol transarterial chemoembolization for hepatocellular carcinoma: a systematic review of efficacy and safety data. *Hepatology*. 2016;64(1):106–116. doi:10.1002/hep.28453
3. Zhou J, Sun H, Wang Z, et al. Guidelines for the diagnosis and treatment of primary liver cancer (2022 Edition). *Liver Cancer*. 2023;12 (5):405–444. doi:10.1159/000530495
4. Finn RS, Qin S, Ikeda M, et al. Atezolizumab plus Bevacizumab in unresectable hepatocellular carcinoma. *N Engl J Med*. 2020;382 (20):1894–1905. doi:10.1056/NEJMoa1915745
5. Qin S, Ren Z, Feng YH, et al. Atezolizumab plus Bevacizumab versus Sorafenib in the Chinese subpopulation with unresectable hepatocellular carcinoma: phase 3 randomized, open-label IMbrave150 Study. *Liver Cancer*. 2021;10(4):296–308. doi:10.1159/000513486
6. Ren Z, Xu J, Bai Y, et al. Sintilimab plus a bevacizumab biosimilar (IBI305) versus sorafenib in unresectable hepatocellular carcinoma (ORIENT-32): a randomised, open-label, Phase 2–3 study. *Lancet Oncol*. 2021;22(7):977–990. doi:10.1016/S1470-2045(21)00252-7
7. Vogel A, Chan SL, Dawson LA, et al. Hepatocellular carcinoma: ESMO Clinical Practice Guideline for diagnosis, treatment and follow-up. *Ann Oncol*. 2025;36(5):491–506. doi:10.1016/j.annonc.2025.02.006
8. Jin ZC, Chen JJ, Zhu XL, et al. Immune checkpoint inhibitors and anti-vascular endothelial growth factor antibody/tyrosine kinase inhibitors with or without transarterial chemoembolization as first-line treatment for advanced hepatocellular carcinoma (CHANCE2201): a target trial emulation study. *EClinicalMedicine*. 2024;72:102622. doi:10.1016/j.eclinm.2024.102622
9. Rumgay H, Arnold M, Ferlay J, et al. Global burden of primary liver cancer in 2020 and predictions to 2040. *J Hepatol*. 2022;77(6):1598–1606. doi:10.1016/j.jhep.2022.08.021
10. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on the management of hepatocellular carcinoma. *J Hepatol*. 2025;82(2):315–374. doi:10.1016/j.jhep.2024.08.028
11. Zhu HD, Li HL, Huang MS, et al. Transarterial chemoembolization with PD-(L)1 inhibitors plus molecular targeted therapies for hepatocellular carcinoma (CHANCE001). *Signal Transduct Target Ther*. 2023;8(1):58. doi:10.1038/s41392-022-01235-0
12. Sayana H, Yousef O, Clarkston WK. Massive upper gastrointestinal hemorrhage due to invasive hepatocellular carcinoma and hepato-gastric fistula. *World J Gastroenterol*. 2013;19(42):7472–7475. doi:10.3748/wjg.v19.i42.7472
13. Grover I, Ahmad N, Googe AB. Hepatogastric Fistula following transcatheter arterial chemoembolization of hepatocellular carcinoma. *Case Rep Gastroenterol*. 2014;8(3):286–290. doi:10.1159/000368302
14. Bale A, Shetty S, Shetty A, Balaraju G, Pai CG. Upper gastrointestinal bleed due to invasive hepatocellular carcinoma and hepato-gastric fistula. *J Clin Exp Hepatol*. 2018;8(1):104–105. doi:10.1016/j.jceh.2017.09.006
15. Liu WY, Ho CC, Wu JM, Chen KH. Laparoscopic closure of gastrohepatic fistula complicated by transarterial chemoembolization for hepatocellular carcinoma. *Asian J Surg*. 2024;47(10):4393–4394. doi:10.1016/j.asjsur.2024.07.168
16. Park YH, Kang SH, Kim SU, et al. A case of hepaticoduodenal fistula development after transarterial chemoembolization in patient with hepatocellular carcinoma. *Korean J Gastroenterol*. 2011;58(3):149–152. doi:10.4166/kjg.2011.58.3.149
17. Prachayakul V, Aswakul P. Spontaneous hepaticoduodenal and choledochoduodenal fistula mimicking duodenal ulcer perforation, a very rare complication of transarterial chemoembolization. *Case Rep Gastroenterol*. 2015;9(1):68–73. doi:10.1159/000369248
18. Wang C, Yang Z, Xia J, Wang W, Chen W, Wang Q. Bronchobiliary fistula after multiple transcatheter arterial chemoembolizations for hepatocellular carcinoma: a case report. *Mol Clin Oncol*. 2018;8(4):600–602. doi:10.3892/mco.2018.1574
19. Fang TK, Huang YN, Chiang TY, Liu XB, Lu YB. Complications of transcatheter arterial chemoembolization for hepatocellular carcinoma: a case report of bronchobiliary fistula development in a 68-year-old man. *Am J Case Rep*. 2023;24:e939195. doi:10.12659/AJCR.939195
20. Li Q, Ao GK, Duan F, Wang ZJ, Yan JY, Wang MQ. Incidence and therapeutic frequency of extrahepatic collateral arteries in transcatheter arterial chemoembolization of hepatocellular carcinoma: experience from 182 patients with survival time more than 3 years. *Eur J Radiol*. 2015;84 (12):2555–2563. doi:10.1016/j.ejrad.2015.10.006
21. Kim HC, Chung JW, Kim WH, et al. Chemoembolization of the left inferior phrenic artery in patients with hepatocellular carcinoma: 9-year single-center experience. *AJR Am J Roentgenol*. 2010;194(4):1124–1130. doi:10.2214/AJR.09.3030
22. Gurses IA, Gayretli O, Kale A, Ozturk A, Usta A, Sahinoglu K. Inferior Phrenic Arteries and Their Branches, Their Anatomy and Possible Clinical Importance: an Experimental Cadaver Study. *Balkan Med J*. 2015;32(2):189–195. doi:10.5152/balkanmedj.2015.150052

23. Morante A, Romano M, Cuomo A, et al. Massive gastric ulceration after transarterial chemoembolization for hepatocellular carcinoma. *Gastrointest Endosc*. 2006;63(4):718–720. doi:10.1016/j.gie.2005.10.021
24. Chennavasin P, Phoopat J, Udomchaisakul P, Gururatsakul M. Delayed gastric perforation following transarterial chemoembolization: case report. *Int J Surg Case Rep*. 2023;104:107965. doi:10.1016/j.ijscr.2023.107965
25. Kim JS, Lee HN, Lee WH, Bae SH. Diaphragmatic perforation after transcatheter arterial chemoembolization of hepatocellular carcinoma via inferior phrenic artery: a case report. *BMC Gastroenterol*. 2022;22(1):46. doi:10.1186/s12876-022-02110-6
26. Chintalapati SP, Patel A, Conjeevaram H. Gastric and duodenal ischaemia after transarterial chemoembolisation for hepatocellular carcinoma: an unexpected but significant complication. *BMJ Case Rep*. 2018;2018:bcr2017223339. doi:10.1136/bcr-2017-223339
27. Kim HC, Chung JW, Lee W, Jae HJ, Park JH. Recognizing extrahepatic collateral vessels that supply hepatocellular carcinoma to avoid complications of transcatheter arterial chemoembolization. *Radiographics*. 2005;25(Suppl 1):S25–39. doi:10.1148/rg.25si055508
28. Cappuccio S, Marinucci B, Bove V, et al. Rare case of endoscopic treatment for bevacizumab-related gastric perforation in a patient with ovarian cancer. *Chemotherapy*. 2020;65(1–2):54–57. doi:10.1159/000508556
29. Chuai Y, Rizzuto I, Zhang X, et al. Vascular endothelial growth factor (VEGF) targeting therapy for persistent, recurrent, or metastatic cervical cancer. *Cochrane Database Syst Rev*. 2021;3(3):CD013348. doi:10.1002/14651858.CD013348.pub2
30. Dias OM, Barbosa CC, Teixeira LR, Vargas FS. Gastropleural fistula from gastric perforation due to renal cell carcinoma after bevacizumab chemotherapy: a case report. *Clinics*. 2011;66(8):1495–1498. doi:10.1590/s1807-59322011000800032
31. Dohrmann T, Kutup A, Mahner S, et al. Gastropleural fistula in a patient with recurrent ovarian cancer receiving combination therapy with Carboplatin, gemcitabine, and bevacizumab. *J Clin Oncol*. 2013;31(12):e208–10. doi:10.1200/JCO.2012.45.1708
32. Fukumura D, Kloepper J, Amoozgar Z, Duda DG, Jain RK. Enhancing cancer immunotherapy using antiangiogenics: opportunities and challenges. *Nat Rev Clin Oncol*. 2018;15(5):325–340. doi:10.1038/nrclinonc.2018.29
33. Rimassa L, Finn RS, Sangro B. Combination immunotherapy for hepatocellular carcinoma. *J Hepatol*. 2023;79(2):506–515. doi:10.1016/j.jhep.2023.03.003
34. Cheng AL, Qin S, Ikeda M, et al. Updated efficacy and safety data from IMbrave150: atezolizumab plus bevacizumab vs. sorafenib for unresectable hepatocellular carcinoma. *J Hepatol*. 2022;76(4):862–873. doi:10.1016/j.jhep.2021.11.030
35. Xia S, Gong H, Wang YK, et al. Pneumocystis jirovecii pneumonia associated with immune checkpoint inhibitors: a systematic literature review of published case reports and disproportionality analysis based on the FAERS database. *Front Pharmacol*. 2023;14:1129730. doi:10.3389/fphar.2023.1129730
36. Cai DH, Fang XL. Pyopneumothorax caused by *Trichomonas tenax* and *Porphyromonas endodontalis* coinfection in a patient with previous cerebral infarction: a case report and literature review. *Infect Drug Resist*. 2022;15:6101–6108. doi:10.2147/IDR.S381859
37. Li Y, Shi P, Zhu R. A pulmonary abscess caused by Porphyromonas endodontalis infection: A case report and literature review. *Diagn Microbiol Infect Dis*. 2024;108(1):116126. doi:10.1016/j.diagmicrobio.2023.116126