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Research Article

Clostridium perfringens liver abscess after transarterial chemoembolization for liver metastatic cancer

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Abstract

Transarterial chemoembolization (TACE) can cause severe complication including liver abscess. Most liver abscess are caused by *E. coli*. However, in our center, *Clostridium perfringens* were identified as a new pathogen in 3 cases, where CT scan showed gas-forming cavity located in the liver. The patients showed obvious symptoms of poisoning, shock, and multiple organ failure. 1 case died soon, and 2 cases survived. The pathogenic understanding, early diagnosis and corresponding treatment of this disease are important to ensure the survival of patients.

Key words: Chemoembolization, Liver cancer; Liver Abscess, *Clostridium perfringens*

Introduction

TACE is an important therapy for both primary and metastatic liver cancer. Despite of its minimum invasion, TACE can cause severe complications. The mortality rates reported due to TACE complications range from 13.3% to 50%.(1) Among complications of TACE, liver abscess formation is one of the severe complications (2-4) with high morbidity and mortality rates. In our center, the incidence of liver abscess after TACE is about 0.84%, which is similar to a previous study by Song et al.(5)

Besides gastrointestinal bacteria, such as *E. coli*, were identified as the main pathogens in liver

abscess, Klebsiella and Staphylococcus were observed from specimens of abscess by cell culture. Recently, we diagnosed liver abscess caused by *Clostridium perfringens* infection after TACE in three liver metastatic cancer patients.

Materials and method.

1. Patients and treatment protocol

From Apr 2015 to Nov 2018, three patients who were diagnosed with advanced gallbladder cancer and bile duct cancer with liver metastases received conventional TACE. Two were male, and one was female (ave. 60.5 years); two cases accepted bile duct resection and biliary-enteric anastomosis; one underwent three sessions of TACE. 1-3 days after TACE, they had high fever and sepsis syndrome and emergency computed tomography (CT) revealed gas-forming cavity in the liver. Post-TACE liver abscess was diagnosed through clinical manifestations, laboratory, and imaging tests.

2. TACE technique

Selective angiography of the celiac artery was performed using a 5 F RH catheter (COOK Corp., USA) inserted through the vascular sheath. Super selectivity was achieved using a microcatheter (Progreat, Terumo Corp., Japan), we injected lipiodol (Guerbet Corp., France) and PVA until stasis was observed in the feeding artery of the tumor. None of them received prophylactic antibiotics before TACE treatment, and conventional hepatoprotective treatments were given for symptomatic relief.

3. Manifestations of liver abscess

24-51 hours after TACE, 3 patients suffered from high fever (≥39°C). They all had chills and one of them had peritonitis manifestations. Blood or pus culture was positive, with *Clostridium perfringens* infection. Based on laboratory and imaging test results, all the cases were diagnosed with liver abscess, located on the right lobe of the liver, confirmed by surgery or imaging tests.

4. Anti-liver abscess treatment:

Two patients underwent fluoroscopy-guided percutaneous transhepatic external drainage. An 8.5-F external drainage tube (COOK Group Co., Ltd., Bloomington, USA) was placed into the abscess cavity. One patient underwent fluoroscopy-guided needle puncture, and another case accepted surgical drainage. The blood and the drainage fluid were subjected to bacterial culture and drug susceptibility tests. These two cases were treated empirical antibiotics and anti-acidosis to prevent liver and renal failure and correct bleeding and thrombocytopenia.

Results

We gave three patients empirical antibiotics with imipenem, anti-acidosis treatment at first, 3-5 days later, when bacterial culture proved the infection were caused by *Clostridium perfringens*; we used imipenem. After percutaneous or surgical drainage and anti-inflammatory treatments, one patient dead 1 week later. 2 patients recovered completely, they survived 12 month and 15 months respectively;

Case 1

A 67-year-old male had accepted a bile duct resection and biliary-enteric anastomosis 6 years ago after post-operative pathology proved well-differentiated adenocarcinoma. He received 4 cycles of systemic chemotherapy. Two years ago, he had a fever and subsequently cancer recurrence

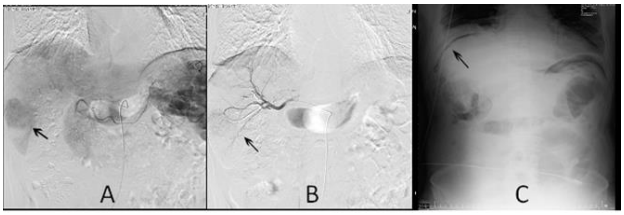

Figure 1A) Angiogranphy from celiac showed tumor stain in S6; B) Post-embolization angiography showed the feeding artery of S6 was completely occluded, tumor stain disappeared; **C**) Plain radiograph demonstrating massive intraabdominal free gas bilaterally in the subdiaphragmatic spaces.

Table 1 Blood examination before and after TACE

Time	WBC	Neut%	Hb	PLT
6 h Pre-TACE	7.45	64.9	141	134
64 h after TACE	3.15	82.34	131	82
78 h after TACE/Pre-op	7.31	88.54	124	61
4 days after TACE	6.73	91.64	97	40
5 days after TACE	13.48	91.74	79	20
6 days after TACE	9.95	85.64	83	17
7 days after TACE	8.07	81.14	87	38
8 days after TACE	9.02	85.24	81	50
9 days after TACE	9.95	87.2	85	75
10 days after TACE	9.13	88.94	81	128

was considered after multiple nodules in liver were found by CT. He received two interventional therapies before he came to our center for the third interventional therapy. When the patient came to our hospital, he had no special clinical sign. He had type II diabetes and was treated with an oral hypoglycemic agent. He had chronic gastric ulcer. He had no history of hepatitis or liver cirrhosis. In physical examination, consciousness was clear, and no scleral jaundice was noted. Laboratory examinations indicated no liver dysfunction and elevation of serum bilirubin, C-reactive protein was negative. WBC count was 7.45×10^9/L, neutrophils 64.9%, PCT was 25.97 ng/ml.

Abdominal enhanced MRI showed: tumor located in S6 enlarged compared with the previous image. After interventional treatment, angiography in celiac artery showed that tumor was stained in segment VI of liver and catheter was advanced into segment VI feeding artery. The patient was treated with 3 ml of lipiodol and 0.2 g of PVA particles with diameter of 100 μm and then returned to ward without any discomfort. [Fig. 1A-B]

24 hours after TACE, he had a fever at 38 ºC and the temperature was controlled by indomethacin suppository; 52 hours after TACE, his temperature raised to 39 ºC. Emergency blood testing showed WBC count was 3.15×10^9/L and neutrophils ratio was 82%. After 3.0 g Sulperazon was given against potential infection, the temperature decreased.

64 hours after TACE, his general conditions became deteriorated: heart rate increased to 120-140 bpm with low oxygen saturation and Gram-negative bacteria were found by blood culture.

Table 2 Biochemical examination before and after TACE

Time	Alb	T-bil	D-bil	ALT	AST	Urea	Crea	UA
6h Pre-TACE	45.5	14.4	4.4	24	26	804	92	413
64h after TACE	38.1	599.7	383.6	583	511	19.53	156	344
78h after TACE/Pre-op	-	-	-	536	370	20.01	180	336
4 days after TACE	26.7	374.8	331.2	291	145	21.49	227	-
5 days after TACE	30.2	290.9	213.5	151	64	-	308	-
7 days after TACE	32.1	126.1	105.3	52	32	-	306	4.13
9 days after TACE	33.8	114	84.9	20	15	35.63	293	4.09
10 days after TACE	33.3	64.6	60.4	16	26	33.69	279	5.82
12 days after TACE	33.8	47.1	42.1	14	15	29.26	227	-

13 days after TACE	37.9	50.9	49.5	14	13	-	218	-

66 hours after TACE, bedside chest and abdominal plain film showed subdiaphragmatic free gas shadow [Fig. 1C]. Serum creatinine was 156 μmol/L and Urea was 19.53 mmol/L. Liver function progressively deteriorated after TACE. AST raised to 511 U/L, ALT 583 U/L, total bilirubin 599.7 μmol/L and direct bilirubin 383.6 μmol/L. He presented fever and deep jaundice. Blood test results were listed in Table 1-2.

68 hours after TACE, gas and ascites in lower abdomen were found by bedside ultrasound, greenish pus was found in left lower abdomen by percutaneous drainage. Smear shows Gram-negative bacteria. Surgeon considered he might be gastrointestinal perforation due to gastric ulcer history, and the patient had septic shock, acute liver failure and acute renal insufficiency and respiratory disorders. The patient was treated with empirical antibiotics (imipenem) and anti-acidosis treatment. In next 10 hours, 1000 ml of fluid was drained from abdominal cavity, and his general conditions turned well. After an emergency abdomen CT, an 83×48 mm area of low attenuation in the posterior segment of the right hepatic lobe with gas formation was found. No sign of enlargement or gas containing in bile duct. No signs of continuous interruption of the bowel. A greater amount of free gas shadow can be seen under the diaphragm, while a small amount of fluid can be seen around margin of liver and spleen. High-density lipiodol can also be observed. [Fig. 2A-C]

It was considered that the patient had abdominal infection, liver abscess, sepsis, septic shock, metabolic acidosis, ARDS, thrombocytopenia and multiple organ dysfunction. 80 hours after TACE, emergency laparotomy and peritoneal drainage was performed. The patient was transferred into ICU with tracheal intubation and mechanical ventilation for respiratory distress. Anti-liver and renal failure, anti-bleeding, anti-acidosis, phlegm and other symptomatic treatment were given. 1 month later, he gradually recovered from ARDS, liver and kidney dysfunction, thrombocytopenia, anemia and hypoproteinemia.

The final blood bacterial culture results showed aerobic bacteria-free growth, *Clostridium perfringens*. 3 weeks later, CT scan showed abscess was absorbed and liver rapture became smaller. [Fig. 2D]

Case 2

A 54-year-old male suffered from obstructive jaundice which was caused by gallbladder lesion who accepted a cholecystectomy and biliary-enteric anastomosis one year ago. Pathology proved to be well-differentiated adenocarcinoma. He had cholangioenterostomy reflux after the surgery and controlled by antibacterial. One month ago, we found multiple liver metastases.

He had no special clinical sign when the he come to our hospital. He had no history of liver cirrhosis. On physical examination, his consciousness was clear and slight jaundice was noted.

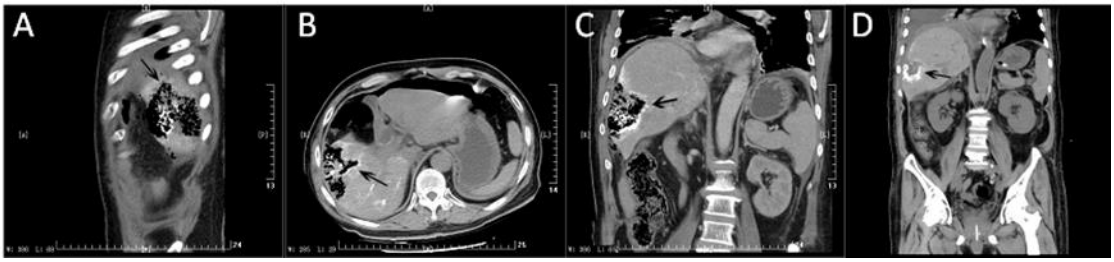

Figure 2A-C) CT found an 83×48 mm area of low attenuation in the posterior segment of the right hepatic lobe with gas formation. No sign of enlargement or gas containing in bile duct; **D**) after 3 weeks, CT scan showed abscess in abdominal cavity was formed and liver rupture turn smaller.

Laboratory examinations indicated Alb was low and serum bilirubin increased slightly, CEA 444.6 (ng/mL), CA199 511.8 U/mL, Albumin 32.3 (g/L), T-bil 27.6 μmol / L, D-bil 20.7 μmol/L, ALT 81 IU/L, AST 107 IU/L,WBC 9.93×10^{12}/L, hemoglobin 114.00 g/L, platelet count 298.00×10^{9}/L, the percentage of neutrophils was 77.01%. C-reactive protein was negative.

We gave him interventional therapy and 8 mL of lipiodol which was mixed with 20 mg of epirubicin for embolization. 24 hours after TACE, he had a fever of 38 ℃. As he accepted embolization, we considered absorption heat. 46 hours after TACE, his temperature raised to 39.5 ℃. The WBC count was 25.38×10^{9}/L, platelet count was 146×10^{9}/L, neutrophil percentage was 91.54%,4 ALT 156 IU/L, AST 268 IU/L, T-bil 40.56 μmol/L. Thus we gave him 2.4 g of procalcitonin.

Emergency CT scan showed irregularly shaped liver nodules and tumors. In the right lobe, there were lipiodol deposition and gas density. The size of largest lesion was about 53×48 mm, intrahepatic bile duct exhibited mild dilatation and part of the bile duct showed gas shadow. No signs of ascites. [Fig. 3A&B]

As he had cholangitis history, the abscess diagnosis was made. Considering he had intractable cholangitis for one-month last year and finally controlled by otapenem, so we used otapenem as empirical antibiotics. His temperature dropped in

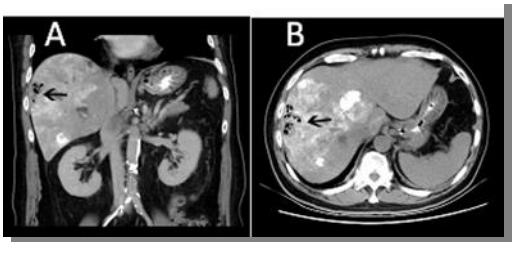

Figure 3A-B): Post-embolization emergency CT scan showed irregularly liver nodules and tumors. In the right lobe, there were gas density.

next day. He refused further examination and drainage because of economic reasons, then he left hospital and move back to county hospital. Follow-up showed he died for shock 1week later, despite of fully dosed antibiotics therapy. The final examination showed WBC was 46.86×10^{9}/L, neut was 95.20%, PLT was 43×10^{9}/L, Glu was 23.4 mmol/L. Blood culture showed *Clostridium perfringens* and *E. coli*.

Discussion

Although pyogenic hepatic abscess after TACE is rare and the reported incidence is less than 0.5%,(5-8) it can cause significant morbidity and mortality. Gas-forming pyogenic liver abscess occurring in 60% of primary liver abscess cases.(9) The cause of liver abscess after TACE is associated with many factors: necrosis forms after tumor tissue embolization combined with chemotherapy-induced liver dysfunction may induces abscess formation.(10) Patients typically have underlying conditions, such as diabetes, malignancy, liver cirrhosis, or an immunosuppressive state.(11-16) Lv et al.(17) claimed that 57.1% of the patients had a medical history of either bilioenteric anastomosis or biliary stent implantation. Lee et al. reported three cases with three dangerous risk factors: diabetes, biliary-enteric anastomosis and liver malignancy.(7) As blood supply of liver cancer usually comes from hepatic artery, during TACE, we usually embolized branch of hepatic artery. However, once TACE blocked the tumor-feeding artery as well as peribiliary capillary plexus, it might cause secondary chemical and hypoxic-ischemic injury to the biliary system.(18) Because the blood supply of the biliary system and peribiliary capillary plexus also originates from the accompanying branch of

the hepatic artery.(19) If bacteria are present in the biliary system at this time, it is easy to result in an infection. When embolic substances remain in the peribiliary capillary plexus, they will not only reduce local blood supply and subsequently weaken the anti-infective ability of the surrounding liver tissues, but also cause microvascular damage of the peribiliary capillary plexus. As a result, the bile leakage can progress to peripheral bile duct necrosis and eventually result in local abscesses and biloma.(20) These three cases all have a history of biliary tract disease or biliary tract surgery, which increases the possibility of bacteria in the biliary tract;

Clostridium perfringens is a Gram-positive, rod-shaped, anaerobic, and spore-forming bacterium of the genus Clostridium .(21) *Clostridium perfringens* is found in the soil and the human gastrointestinal. It is a part of the intestinal normal flora in humans and known to cause tissue necrosis and gas gangrene by producing α-toxin. Because *Clostridium perfringens* infection is a type of severe infections, the time remaining for patient is normally very limited. Early diagnosis is difficult because only nonspecific inflammation and gas formation in the focus are present. However, once α-toxin triggers hemolysis, it progresses very rapidly followed by acidosis and renal failure.(22, 23)Van Bunderen(24) reported, among 11 cases of *Clostridium perfringens* septicemia with liver abscess, 10 had died in 90.9%. Over half of the patients presented elevated bilirubin and LDH as well as anemia, suggesting hemolysis at the initial presentation.

Liver abscess caused by *Clostridium perfringens* after TACE has been reported,(25) which can result in massive hemolysis and death within several hours. Gas-forming pyogenic liver abscess also ruptures easily because of tissue invasion and fragility of abscess wall, and further gas formation increases the internal pressure of the abscess. In previous studies, spontaneous rupture of liver abscess has occurred in 7.16-15.1% of patients.(26, 27) Besides antibiotic, urgent removal of the focus and antibiotic administration are essential for survival, control of hemolysis and supportive care including hemodialysis are all necessary.

Our reported case all presented gas-forming pyogenic liver abscess, which developed septic shock with multi-organ system failure. Usually, percutaneous catheter drainage is recommended as a first-line treatment with a 95.2% curative rate.(28) The first case accepted percutaneous catheter drainage and surgical drainage, while the second case gave up further treatment and died very soon.

What lessons could be learned from these three cases?

When gas-forming abscess with severe symptom was found, possibility of *Clostridium perfringens* infection should be considered. In first case, a wrong judgement was made, while, in the second case, *Clostridium perfringens* was tested, which helped us to take the right procedure.

For the first case, when the subdiaphragm free gas was found, perforation of the digestive tract was considered due to his gastric ulcer history,(29) the gas in right hepatic lobe area was noticed, but colonic gas was considered; after CT scan, we identified liver from transverse colon liver curvature colon and made the right diagnosis. So, if possible, a CT scan is necessary to reveal more details. High bilirubin was another reason for misjudgment, in which it was induced by obstructive jaundice or side effects of embolization. However, bile duct dilatation was not observed on CT and therefore obstructive jaundice was excluded; Another possible reason was the damage of the toxin produced by *Clostridium perfringens* to the liver cell. The bilirubin decreased after the infection was controlled.

Since bilirubin decreased dramatically in 3 days after surgery, there may be another possible reason:

the infection was from bile duct. When the liver raptured, bilirubin in bile duct could flow into the abdominal cavity through the rupture site, and then absorbed by the omentum. In obstructive jaundice, the bilirubin couldn't flow into intestinal and enter the blood circulation. However, in this case, the mechanism is somewhat like bile duct-greater omentum shunt, where majority bilirubin flow into the abdominal cavity directly through raptured liver, similar to what happened when gallbladder rupture(30). When we drained necrosis tissue and exudate from abdominal cavity, blood bilirubin level decreased quickly. With the liver regeneration and repair of rupture site, the bile leakage reduced gradually, and total bilirubin and direct bilirubin also returned to normal level.

Conclusion

Liver abscess caused by *Clostridium perfringens* is very rare and insidious, it is more serious and more deadly than the liver abscess caused by other bacteria. Early surgical drainage could provide more opportunities for survival. If the patient has obvious poisoning symptoms and the CT shows a gas-containing cavity, the disease should be considered and given anti-sepsis as early as possible. Good drainage and suitable antibacterial therapy may help patients recover earlier.

Declarations

1) ***Consent to publication***
We declare that all authors agreed to publish the manuscript at this journal based on the signed Copyright Transfer Agreement, and followed publication ethics.

2) ***Ethical approval and consent to participants***
Not applicable.

3) ***Disclosure of conflict of interests***
We declare that no conflict of interest exists.

4) ***Funding***
None

5) ***Availability of data and material***
We declare that the data supporting the results reported in the article are available in the published article.

6) ***Authors' Contributions***
Authors contributed to this paper with the design (ZLZ), literature search (ZLZ), data collection (all other authors), drafting (all other authors), revision (ZLZ and all other authors), editing (ZLZ) and final approval (ZLZ).

7) ***Acknowledgement***
None

8) ***Authors' biography***
None

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