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Concurrent alcoholic cirrhosis and malignant peritoneal mesothelioma in a patient: A case report

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Abstract

BACKGROUND

Malignant peritoneal mesothelioma (MPM) originates from the mesothelial and subcutaneous cells of the abdominal cavity. It is difficult to diagnose due to its nonspecific and vague symptoms and should be differentiated from alcoholic cirrhosis, liver cancer and pancreatic cancer. Misdiagnosis and missed diagnosis can easily occur when MPM is combined with other diseases. At present, there is no case report of the MPM concurrent with alcoholic cirrhosis.

CASE SUMMARY

A 63-year-old man presented to our hospital with abdominal distension for 20days. He had a history of alcohol consumption for nearly 30 years and no history of special drug use or toxic exposure. After a period of treatments for alcoholic cirrhosis in a community hospital, his symptoms did not improve significantly. The patient underwent exploratory laparotomy and surgical resection in our hospital. Pathologic examination showed an epithelioid MPM. He was treated with chemotherapy and intraperitoneal hyperthermic perfusion after surgery. He is now in stable condition with no tumor recurrence.

CONCLUSION

Misdiagnosis and missed diagnosis of MPM can easily occur because of its insidious onset. We should deepen our understanding of the MPM in clinical practice, make correct diagnosis and provide timely and effective treatments.

INTRODUCTION

Malignant peritoneal mesothelioma (MPM) is a rare malignancy originated from peritoneal epithelium or mesothelium and the annual incidence of the tumor in the general population is 1-2 cases per million. It was first reported by Miller J in 1908[1]. In recent years, studies have shown that the incidence of the MPM in asbestos exposure is significantly higher than that in non-asbestos exposure[2]. Pathological together with immunohistochemical examination is the gold standard for its diagnosis. MPM has a hidden onset and the clinical symptoms of patients are not typical, so missed diagnosis and misdiagnosis may occur. We here report a case where the clinical manifestations such as abdominal distension and ascites and abdominal imaging findings were consistent with alcoholic cirrhosis, which made clinicians miss the diagnosis of the MPM.

CASE PRESENTATION

Chief complaints

A 63-year-old man presented to our hospital with a history of abdominal distension for 20 days.

History of present illness

The patient's abdominal distension was persistent and worsened after meals. He vomited an average of 1-2 times per day, with no coffee-like contents. He was diagnosed with multiple hepatic cysts, alcoholic cirrhosis and ascites by abdominal computed tomography (CT) in a community hospital. After a period of

treatments according to the plan for alcoholic cirrhosis, the patient's symptoms did not improve significantly.

History of past illness

The patient had a history of schizophrenia for many years, he denied a history of other diseases and surgery. The patient also had a history of alcohol consumption for nearly 30 years and with no history of special drug use or toxic exposure. He was diagnosed as hepatic cirrhosis by liver biopsy 3 years ago.

Personal and family history

He denies any personal and family history.

Physical examination

Physical examination on admission showed abdominal distension, full abdominal tenderness and positive movement dullness, but no splenomegaly was observed.

Laboratory examinations

Laboratory tests results were as follows: white blood cell count 7.59 × 10^9/L, Platelet count 507 × 10^9/L, hemoglobin level 105 g/L, C-reactive protein 32.29 mg/L, erythrocyte sedimentation 51 mm/h, procalcitonin 0.82 ng/mL, albumin 33.9 g/L and D-dimer level of 2.15 mg/L. Exudate was found in the examination of ascites and the serum ascites albumin gradient (SAAG) level of 9.2g/L. Results of other laboratory tests, including carcinoembryonic antigen (CEA), alpha fetoprotein (AFP), carbohydrate Antigen 125 (CA125), carbohydrate antigen 19-9 (CA199), carbohydrate Antigen 50 (CA50), antin-uclear antibody, anti-mitochondrial antibody, anti dsDNA antibody, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transpeptidase (GGT), coagulation function, hepatitis B surface antigen and hepatitis C antibody were unremarkable. Malignant tumor cells were found in the exfoliated cells of ascites.

Imaging examinations

Contrast-enhanced CT scan confirmed the findings of the CT scan at the community hospital (**Figure 1)**. More importantly, a large amount of fluid was observed in the abdominal cavity and the right peritoneum was irregularly thickened with nodular thickening of the greater omentum (**Figure 2**).

FINAL DIAGNOSIS

The patient then underwent exploratory laparotomy. A large amount of yellowish ascites was found in the abdominal cavity. There were extensive adhesions between the diaphragm, stomach, spleen and abdominal wall of the liver. Adhesions were severe in most of the small intestine and the mesentery was contracted. The greater omentum was pancake-shaped, multiple round masses were observed in the parietal and visceral peritoneum, with a diameter of 3mm–20mm and the right subphrenic peritoneum was thickened obviously, with an area of about 15 × 15 cm (**Figure 3**). Tumors over 3mm, partial right subphrenic peritoneum and the greater omentum were resected. Pathologic examination showed an epithelioid MPM (**Figure 4**).

TREATMENT

In addition to general comprehensive treatments, the patient was administered pemetrexed in combination with cisplatin and intraperitoneal hyperthermic perfusion after surgery.

OUTCOME AND FOLLOW-UP

At present, we have followed up for 11 mo and he is in stable condition with no tumor recurrence.

DISCUSSION

MPM is a malignant tumor originating from mesothelial and subcutaneous cells of the abdominal cavity. Histologically, there are epithelioid, sarcomatoid and biphasic types[3]. There was no significant difference in the degree of malignancy among different types. Elderly men are among the high-risk group for MPM. Recently, cases of MPM in young adults have also been reported[4]. MPM is a rare entity and has been linked to industrial pollutants and mineral exposure. The increase diffusion of chemicals and a steep rise of cancer incidence is an established fact. For MPM, the most common carcinogen is asbestos, approximately 80% of cases were associated with asbestos exposure[5,6]. The pathogenesis of MPM is unknown. *BAP1* mutation had been revealed in some patients with MPM by gene analyses, but it was not the only gene involving inherited predisposition to the MPM[7]. MPM has insidious onset, and the most common initial symptoms of the MPM are abdominal pain, abdominal distension, significant weight loss, ascites, anorexia and night sweat[8]. Some patients have concurrent paraneoplastic syndromes associated with MPM, such as hypoglycemia, thrombocytosis, venous thrombosis, paraneoplastic liver disease and wasting syndrome[8]. MPM is difficult to diagnose due to its nonspecific and vague symptoms and should be differentiated from alcoholic cirrhosis, liver cancer and pancreatic cancer. Serological tests and tumor markers are of little value. There is no specific imaging technique for MPM. Currently, CT, especially the contrast-enhanced CT, is widely used. Extensive and irregular thickening of peritoneum, mesentery and omentum accompanied by massive peritoneal effusion are typical manifestations of MPM in CT scan. Positron emission tomography (PET-CT) is valuable in early diagnosis, evaluation of curative effect and judgment of distant metastasis. The prognosis of patients with MPM is poor. Tumor resection or palliative resection is preferred in the early stage. Pemetrexed combined with cisplatin is a widely accepted chemotherapy for inoperable patients. More clinical trials are needed for the promising treatment of immunotherapy and targeted therapy[9].

In the present case, the patient had a history of long-term alcohol consumption, and the CT sanning showed cirrhosis and ascites, which resulted in a diagnosis of alcoholic

cirrhosis in the community hospital. After the patient was transferred to our hospital due to poor treatment effect, we performed the contrast-enhanced CT examination and ascites cytology, which confirmed the diagnosis of MPM.

Therefore, a correct diagnosis of rare diseases including MPM, is always necessary so to adjust the treatments plan timely.

There were some limitations in this report. First, relationships between alcoholic cirrhosis and MPM are unknown, further studies are needed to determine whether there is a correlation between the two diseases. Second, a long-term follow-up remains necessary.

CONCLUSION

MPM is subjected to misdiagnosis and missed diagnosis because of its insidious onset. Clinicians should be aware if the disease, and make a correct diagnosis so as to provide patients with timely and effective treatment. At the same time, further research on the pathogenesis of the MPM is urgently needed.

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