Misdiagnosed as pancreatic cancer seven years ago, a 29-year-old woman suffered from left-sided portal hypertension caused by peripancreatic lymph node tuberculosis: a case report and literature review

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Case report

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Abstract

Background: As the only curable portal hypertension, left-sided portal hypertension (LSPH) is a very rare clinical syndrome. With normal liver function, LSPH is mostly due to pancreatic disease and its complications that cause spleen vein compression, inflammatory wall thickening or lumen blockage, isolated splenic vein thrombosis, restricted splenic vein reflux, finally resulting in increased splenic vein pressure, opened collateral circulation, and bleeding from isolated gastric varices. With a quiet occurrence, LSPH often lacks specific symptoms, which finally leads to difficult diagnosis. Therefore, acuminous options of clinical examination are exceedingly crucial. Splenectomy is the prime treatment for cases complicated by variceal bleeding, but the effect of treatment depends mainly on the condition of the primary disease. Other than these, diseases resulting in LSPH often need to be distinguished from pancreatic cancer, so it is necessary for us to pay more attention to the diagnosis and treatment of LSPH.

Case presentation: Here, we report a case of 29-year-old women who was admitted to the hospital for repeated hematemesis and black stool, with a differential diagnosis of pancreatic cancer seven years ago. Abdominal computed tomography (CT), CT angiography (CTA), portal phase three-dimensional vascular reconstruction, and gastroscopy indicated varicose gastric fundus veins, pancreatic mass, and enlarged peripancreatic lymph nodes. Erythrocyte, platelet, and leukocyte counts in decline, positive gamma interferon release assay, and normal liver function were given by laboratory examination. Abdominal exploration, splenectomy, varicose veins dissection, and lesions resection were performed by laparotomy. After surgery, the diagnosis of lymph node tuberculosis was confirmed by the technology of biopsy. Based on mention above, a diagnosis of LSPH caused by peripancreatic lymph node tuberculosis was confirmed. Postoperative evolution was steady, and the patient was in ideal clinical status at 3 months follow-up.

Conclusions: We reported the first case of LSHP caused by peripancreatic lymph node tuberculosis. At the same time of resulting in left portal hypertension, the peripancreatic lymph node tuberculosis is often misdiagnosed as pancreatic cancer. Further studies were necessary to explore more favorable diagnosis method for pancreas mass and more advantageous therapy for LSPH, especially caused by mechanical compression.

Background

Lymph nodes are areas most frequently affected by *Mycobacterium tuberculosis* outside the lung[1–4]. However, intra-abdominal lymph node tuberculosis is a very rare disease, and is associated in most cases with immunodepression[5]. Left-sided portal hypertension is a rare clinical syndrome which may lead to bleeding from isolated gastric varices with a normal liver function[6]. The most common causes of LSPH are chronic pancreatitis, pancreatic pseudocysts and various pancreatic tumors[7–12]. Most patients with left-sided portal hypertension are asymptomatic, and only a few cases present isolated gastric varices, ruptures, and fatal bleeding caused by splenic vein obstruction resulting from thrombosis, mechanical compression, tumor invasion and metastasis[7–14]. It is difficult to diagnose LSPH[15], and

bleeding from LSPH are frequently fatal[8, 13, 16–19], other than these, diseases resulting in LSPH often need to be distinguished from pancreatic cancer, so it is necessary for us to pay more attention to the diagnosis and treatment of LSPH[20–22]. All in all, we reported a case with LSPH caused by peripancreatic lymph node tuberculosis and misdiagnosed as pancreatic cancer seven years before.

Case Presentation

The women underwent debridement and drainage for cervical lymph node tuberculosis nine years ago. Seven years ago, because of abdominal pain, the patient was examined by abdominal computed tomography (CT) in other hospital, which reported that there was a mass in pancreas body with enlarged lymph nodes in abdominal cavity. Naturally, the 29-year-old women was suspected as pancreatic cancer with lymphatic metastasis. However, except CT report, the specific clinical examination index of the patient in other hospital was unavailable. Two years after symptoms relief by treatment of Chinese traditional medicine, the patient showed repeated vomiting and melena for 5 years. In our hospital, the patient stated that her mental, appetite and sleep was normal, with ochrodermia, but without fever, jaundice, petechiae, and ecchymoses. Physical examination showed a good general condition without abdominal tenderness, abdominal muscle tension, rebound pain, abdominal mass, and hepatomegaly. Additionally, there were no swelling in patient's cervical, supraclavicular, axillary and inguinal lymph nodes.

To clarify the cause, the blood biochemical and blood routine examination were performed. The blood biochemical results reported adenosine deaminase (ADA) and liver function within a normal range, the detailed blood biochemical results were shown in Table 1. The blood routine examination indicated erythrocyte, platelet, and leukocyte counts in decline, other blood routine results were reported in Table 2. In addition, the alpha fetal protein (AFP), tumor associated antigen 125 (CA125), and tumor associated antigen 199 (CA199) were normal. Tests for infection with human immunodeficiency virus, hepatitis B virus, hepatitis C virus, and mycobacterium tuberculosis were negative, but gamma interferon release assay was positive.

In order to further diagnose, imaging examinations were performed by us, which including abdominal non-contrast and contrast-enhanced CT, CT angiography (CTA), CT venography (CTV), and portal phase three-dimensional vascular reconstruction. Non-contrast and contrast-enhanced CT indicated that not obviously enhanced in both arterial and venous phases, a heterogeneous, no vascular, and low-density mass is located in pancreas body, with poorly defined edges and a 3.1×2.0 cm diameter (Figure 1A). An intumescent spleen and multiple nodular dense shadows around pancreas, hepatic hilar region, and mesenteric were also detected by CT (Figure 1A). On CTA, CTV, and portal phase three-dimensional vascular reconstruction graphy, enlarged splenic vein, narrowed start section of the splenic vein, and tortuous gastric veins were observed (Figure B and Figure D). Furthermore, gastroscopy detected that there are varicose veins under the gastric fundus mucosa (Figure C), but the esophageal mucosa is smooth. To investigate whether the patient has a history of tuberculosis, chest CT scan was operated, which showed cabled-like and flaky increased density shadows in the posterior segment of the upper lobe

tip of the left lung. Based on the above results, the patient was diagnosed as left-sided portal hypertension. However, nature of the mass in pancreas was not clear, tuberculosis, tumor, or others?

Due to repeated hematemesis symptom, after blood transfusion and anemia symptoms improving, the patient received splenectomy and perigastric fundus vascular dissection. Meanwhile, we operated lesion excision for biopsy. In operating field an enlarged spleen with a 34 × 25 × 15 cm diameter was observed. After dissociating peritoneal adipose tissue, we caught up with that the left gastric vein, right gastric vein, left gastroepiploic vein, and right gastroepiploic vein are extensively tortuous and dilated (Figure 2A). As the deepening dissection, we could see swelling lymph nodes at greater curvature, hepatoduodenal ligament, and lower margin of pancreas (Figure 2B). We considered the mass occupying the lower margin of the pancreas to be an abscess by intraoperative ultrasound. Expressing a caseous necrosis profile, the sample was submitted to biopsy, and a necrotizing granulomatous lymphadenitis compatible with tuberculosis was observed (Figure 2C and 2D). After these results, a microbiological study was also performed, obtaining a suspiciously positive Ziehl-Neelsen staining. Therefore, the diagnosis of left-sided portal hypertension caused by lymph node tuberculosis was confirmed. According to the current clinical evidences, we inferred that the diagnosis of patient seven years ago should be peripancreatic lymph node tuberculosis rather than pancreatic cancer.

Discussion And Conclusions

Tuberculosis is a common pathology in undeveloped countries. However, due to increasing immigration and cases of HIV immunosuppression, its presence is increasing in developed countries in recent years[23]. Pulmonary tuberculosis is the most frequent form of presentation, but celiac lymph node forms are uncommon[24–26]. In this article, we reported a female patient who suffered from left-sided portal hypertension resulting from peripancreatic lymph node tuberculosis, misdiagnosed as pancreatic cancer seven years ago in other hospital. Lacking specific symptoms, the patients with peripancreatic lymph node tuberculosis mainly express abdominal pain, constitutional syndrome, jaundice, emaciation, and pancreatitis or abdominal mass, which are similar to pancreatic cancer. Furthermore, there is a high similarity between peripancreatic lymph node tuberculosis and pancreatic cancer in radiological manifestations[20, 25, 27–30]. Due to all of these factors, peripancreatic lymph node tuberculosis is frequently misdiagnosed as pancreatic cancer. However, the therapeutic approaches of them are totally different, so it is significant to make a correct diagnosis for avoiding unnecessary surgeries and long-term complications. Due to this reason, endoscopic ultrasound-guided fine-needle aspiration biopsy (EUS-FNAB) was utilized to distinguish peripancreatic lymph node tuberculosis from pancreatic cancer by Ziehl-Neelsen staining and mycobacterium tuberculosis culture[30–35]. However, the accuracy of these tests also depends on the quality of sample and the activity of mycobacterium tuberculosis. Hence, surgery could be a favorable way to obtain a certain diagnosis, as in our case, and laparoscopic surgery may be a better option if surgery is just for diagnosis. Moreover, tuberculosis history, ADA, gamma interferon release assay, and the polymerase chain reaction (PCR) of tuberculous bacillus can serve as an important reference for the diagnosis of lymph node tuberculosis[3, 36].

Possessing a normal liver function, patients with LSPH mainly result from splenic vein obstruction, and pancreatic inflammatory or neoplastic diseases are the main causes of LSPH[6]. In our case, swollen lymph node compression or fibrous scarring after caseous necrosis should be the main cause. The diagnosis of LSPH is based on clinical, biochemical, and radiological evaluation. Many patients with LSPH are asymptomatic or have primary disease symptoms[13, 14, 37]. However, there are few LSPH patients expressing isolated gastric venous bleeding and anemia, usually the bleeding is serious[6, 9, 12]. Routine blood tests can show a decrease in red blood cells, lymphocytes, and platelets. Biochemical evaluation is mainly used to exclude cirrhotic portal hypertension and look for primary disease. In addition to clinical symptoms, imaging test plays an important role in confirming the diagnosis in the majority of cases[38]. Angiography of splenic vein remains the gold standard in diagnosing LSPH, but that method is rarely used today because it is invasive and has potential morbidity[39]. Transabdominal ultrasonography (US) is often the initial imaging modality utilized, but the accuracy of trans-abdominal US is limited in detecting splenic or superior mesenteric veins thrombosis[40]. Recently, endoscopic ultrasound (EUS) has been utilized to evaluate the portal vasculature. This technology has a more accuracy than transabdominal US for evaluating patency of the splenic vein[13, 41]. Of course, with fast development of CT and endoscope, combination of multidetector CTA, gastroscope, and portal phase three-dimensional vascular reconstruction may be a better option, as our case report. More importantly, the results of CTA and portal phase three-dimensional vascular reconstruction are competent in guiding the operation, if necessary. Magnetic resonance angiography (MRA) is a promising method for the evaluation of the portal venous system too[42]. In term of treatment, asymptomatic patients whether need treatment remains controversial, but it is necessary to intervene for patients with active bleeding. Besides improving bleeding, it is often necessary to treat the primary disease[16, 17, 19, 43, 44]. According to patient's clinical condition, there are several methods to relieve isolated gastric bleeding. Enjoying ability of reducing venous blood reflux, splenectomy remains the preferred treatment for patients with gastric bleeding due to left portal hypertension, and splenic artery embolization can be used as a supplement for patients who are not suitable for splenectomy[13, 16, 17, 19, 43–45]. Endoscopic therapy has great advantages in the treatment of acute massive gastric bleeding, but the rebleeding is unavoidable[46–49]. We are also considering whether left portal hypertension due to mechanical compression can be corrected by stent implantation, which will be our future research direction. In our therapeutic procedure, splenectomy and varicose veins dissection were performed to intervene hypersplenism and severe varices. In summary, the treatment of patients with left portal hypertension should be tailored to maximize the benefit of patients.

In conclusion, we state a very interesting case. Experiencing a misdiagnosis, a 29-year-old woman suffered from LSPH resulting from peripancreatic lymph node tuberculosis. LSPH is a very rare clinical syndrome, and we reported the first case of LSHP caused by peripancreatic lymph node tuberculosis. For us, it is of necessity to introduce this case to provide reference for clinical diagnosis and treatment of LSPH.

List Of Abbreviations

LSPH: left-sided portal hypertension

CT: computed tomography

CTA: CT angiography

CTV: CT venography

ADA: adenosine deaminase

AFP: alpha fetal protein

CA125: tumor associated antigen 125

CA199: tumor associated antigen 199

EUS-FNAB: endoscopic ultrasound-guided fine-needle aspiration biopsy

PCR: polymerase chain reaction

US: ultrasonography

EUS: endoscopic ultrasound

MRA: magnetic resonance angiography

Declarations

Ethics approval and consent to participate: Not needed as this case is a case admitted in the hospital for treatment and diagnosis.

Consent for publication: Written informed consent for publication of their clinical details and/or clinical images was obtained from patient herself.

Availability of data and materials: Not applicable.

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

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Authors' contributions: DY and JH wrote the manuscript. JG and JL conceived the report. XL and FX accumulated the clinical materials. All authors contributed to the critical revision of the report for important intellectual content. All authors read and approved the final manuscript.

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Footnotes: Corresponding author. #Dajun Yu and Xiaolan Li contribute to this work equally.

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Tables

Table 1. blood biochemical results			
Variable	Result	Units	Reference
Total bilirubin	9.4	umol/L	0.0-22.3
Direct bilirubin	7.8	umol/L	0.0-8.6
Indirect bilirubin	1.6	umol/L	0.0-17.1
Serum total bile acid	10.5	umol/L	0.0-10.0
Alanine aminotransferase	21.0	U/L	5.0-40.0
Aspartate aminotransferase	25.0	U/L	5.0-40.0
Alkaline phosphatase	50	U/L	45-125
Glutamyl transpeptidase	11	U/L	5-60
Lactate dehydrogenase	209	U/L	100-350
Cholinesterase	4426	U/L	3930-1380
Adenosine deaminase	10.0	U/L	4.0-22.0
Alpha-l-fucosidase	26.0	U/L	0.0-40.0
Monoamine oxidase	3.0	U/L	0.0-12.0
Prealbumin	181	mg/L	150-380
Total protein	65.8	g/L	65.0-85.0
Albumin	44.4	g/L	40.0-55.0
Globin	21.4	g/L	20.0-40.0
Albumin/Globin	2.07		1.20-2.40

Table 2. blood routine examination			
Variable	Results	Units	Reference
White blood cells	1.81	*10~9/L	3.50-9.50
Red blood cells	2.60	*10~12/L	3.80-5.10
Hemoglobin	67	g/L	115-150
Platelets	50.00	*10~9/L	85.00-350.00
Hematocrit	23.5	%	35.0-45.0
Mean corpuscular volume	90.40	fL	82.00-100.00
Mean corpuscular hemoglobin	25.80	pg	27.00-34.00
Mean corpuscular-hemoglobin concentration	285.000	g/L	316.00-354.00
Red cell volume distribution width-CV	17.90		0.00-15.00
Red cell volume distribution width-SD	59.10		0.00-45.00
Neutrophil ratio	77.90	%	40.00-75.00
Lymphocyte ratio	15.50	%	20.00-50.00
Monocyte ratio	5.50	%	3.00-10.00
Eosinophil ratio	1.10	%	0.40-8.00
Basophil ratio	0.00	%	0.00-1.00
Neutrophils	1.41	*10~9/L	1.80-6.30
Lymphocytes	0.28	*10~9/L	1.10-3.20
Monocytes	0.10	*10~9/L	0.10-0.60
Eosinophils	0.02	*10~9/L	0.02-0.52
Basophil	0.00	*10~9/L	0.00-0.06

Figures

Figure 1

Images from CT and gastroscop. (A)Non-contrast CT imaging on horizontal plane: There is a low-density and heterogeneous mass in pancreas body with an irregular edge (red arrow). Spleen is enlarged (blue arrow). Nodular dense shadows around pancreas (green arrow). (B)An CTA image at portal venous phase is obtained in a coronal plane. Varicose gastric veins (red arrow). Swollen and calcified lymph nodes (blue arrow). The start section of splenic vein become narrow (green arrow). (C)Varicose vein in gastric fundus is showed by gastroscop. (D)Varicose gastric veins and normal esophageal veins portal are displayed in phase three-dimensional vascular reconstruction image.

Figure 2

Intraoperative circumstance and specimens. (A) Varicose gastric veins (blue arrow). (B) A enlarged peripancreatic lymph nodes (blue arrow). (C) Macroscopic aspect of the swelling peripancreatic lymph nodes. The profile of the swelling peripancreatic lymph nodes present caseous necrosis (blue arrow). (D) No structural necrosis in the background of lymphocytes is showed in hematoxylin-eosin staining.