World J Clin Cases 2022 September 16; 10(26): 9180-9549





#### **Contents**

Thrice Monthly Volume 10 Number 26 September 16, 2022

#### **REVIEW**

Assisting individuals with diabetes in the COVID-19 pandemic period: Examining the role of religious 9180 factors and faith communities

Eseadi C, Ossai OV, Onyishi CN, Ilechukwu LC

9192 Role of octreotide in small bowel bleeding

Khedr A, Mahmoud EE, Attallah N, Mir M, Boike S, Rauf I, Jama AB, Mushtaq H, Surani S, Khan SA

#### **MINIREVIEWS**

9207 Internet of things-based health monitoring system for early detection of cardiovascular events during COVID-19 pandemic

Dami S

9219 Convergence mechanism of mindfulness intervention in treating attention deficit hyperactivity disorder: Clues from current evidence

Xu XP, Wang W, Wan S, Xiao CF

9228 Clinical presentation, management, screening and surveillance for colorectal cancer during the COVID-19 pandemic

Akbulut S, Hargura AS, Garzali IU, Aloun A, Colak C

Early diagnostic value of liver stiffness measurement in hepatic sinusoidal obstruction syndrome induced 9241 by hematopoietic stem cell transplantation

Tan YW, Shi YC

#### **ORIGINAL ARTICLE**

#### **Case Control Study**

9254 Local inflammatory response to gastroesophageal reflux: Association of gene expression of inflammatory cytokines with esophageal multichannel intraluminal impedance-pH data

Morozov S, Sentsova T

#### **Retrospective Study**

Evaluation of high-risk factors and the diagnostic value of alpha-fetoprotein in the stratification of primary 9264

Jiao HB, Wang W, Guo MN, Su YL, Pang DQ, Wang BL, Shi J, Wu JH

One-half layer pancreaticojejunostomy with the rear wall of the pancreas reinforced: A valuable 9276 anastomosis technique

Wei JP, Tai S, Su ZL

#### Contents

#### Thrice Monthly Volume 10 Number 26 September 16, 2022

9285 Development and validation of an epithelial-mesenchymal transition-related gene signature for predicting prognosis

Zhou DH, Du QC, Fu Z, Wang XY, Zhou L, Wang J, Hu CK, Liu S, Li JM, Ma ML, Yu H

#### **Observational Study**

9303 Incidence and risk factor analysis for swelling after apical microsurgery

Bi C, Xia SQ, Zhu YC, Lian XZ, Hu LJ, Rao CX, Jin HB, Shang XD, Jin FF, Li JY, Zheng P, Wang SH

#### **CASE REPORT**

9310 Acute carotid stent thrombosis: A case report and literature review

Zhang JB, Fan XQ, Chen J, Liu P, Ye ZD

9318 Congenital ovarian anomaly manifesting as extra tissue connection between the two ovaries: A case report

Choi MG, Kim JW, Kim YH, Kim AM, Kim TY, Ryu HK

Cefoperazone-sulbactam and ornidazole for Gardnerella vaginalis bloodstream infection after cesarean 9323

section: A case report

Mu Y, Li JJ, Wu X, Zhou XF, Tang L, Zhou Q

9332 Early-onset ophthalmoplegia, cervical dyskinesia, and lower extremity weakness due to partial deletion of

chromosome 16: A case report

Xu M, Jiang J, He Y, Gu WY, Jin B

9340 Posterior mediastinal extralobar pulmonary sequestration misdiagnosed as a neurogenic tumor: A case

report

Jin HJ, Yu Y, He W, Han Y

9348 Unexpected difficult airway due to severe upper tracheal distortion: A case report

Zhou JW, Wang CG, Chen G, Zhou YF, Ding JF, Zhang JW

9354 Special epithelioid trophoblastic tumor: A case report

Wang YN, Dong Y, Wang L, Chen YH, Hu HY, Guo J, Sun L

9361 Intrahepatic multicystic biliary hamartoma: A case report

Wang CY, Shi FY, Huang WF, Tang Y, Li T, He GL

9368 ST-segment elevation myocardial infarction in Kawasaki disease: A case report and review of literature

Lee J, Seo J, Shin YH, Jang AY, Suh SY

9378 Bilateral hypocalcaemic cataracts due to idiopathic parathyroid insufficiency: A case report

Li Y

9384 Single organ hepatic artery vasculitis as an unusual cause of epigastric pain: A case report

Kaviani R, Farrell J, Dehghan N, Moosavi S

9390 Congenital lipoid adrenal hyperplasia with Graves' disease: A case report

Wang YJ, Liu C, Xing C, Zhang L, Xu WF, Wang HY, Wang FT

#### Contents

#### Thrice Monthly Volume 10 Number 26 September 16, 2022

9398 Cytokine release syndrome complicated with rhabdomyolysis after chimeric antigen receptor T-cell therapy: A case report

Zhang L, Chen W, Wang XM, Zhang SQ

9404 Antiphospholipid syndrome with renal and splenic infarction after blunt trauma: A case report

Lee NA, Jeong ES, Jang HS, Park YC, Kang JH, Kim JC, Jo YG

9411 Uncontrolled high blood pressure under total intravenous anesthesia with propofol and remifentanil: A case report

Jang MJ, Kim JH, Jeong HJ

9417 Noncirrhotic portal hypertension due to peripheral T-cell lymphoma, not otherwise specified: A case report

Wu MM, Fu WJ, Wu J, Zhu LL, Niu T, Yang R, Yao J, Lu Q, Liao XY

9428 Resumption of school after lockdown in COVID-19 pandemic: Three case reports

Wang KJ, Cao Y, Gao CY, Song ZQ, Zeng M, Gong HL, Wen J, Xiao S

9434 Complete recovery from segmental zoster paresis confirmed by magnetic resonance imaging: A case report

Park J, Lee W, Lim Y

9440 Imaging findings of immunoglobin G4-related hypophysitis: A case report

Lv K, Cao X, Geng DY, Zhang J

9447 Systemic lupus erythematosus presenting with progressive massive ascites and CA-125 elevation indicating Tjalma syndrome? A case report

Wang JD, Yang YF, Zhang XF, Huang J

9454 Locally advanced cervical rhabdomyosarcoma in adults: A case report

Xu LJ, Cai J, Huang BX, Dong WH

9462 Rapid progressive vaccine-induced immune thrombotic thrombocytopenia with cerebral venous thrombosis after ChAdOx1 nCoV-19 (AZD1222) vaccination: A case report

Jiang SK, Chen WL, Chien C, Pan CS, Tsai ST

9470 Burkitt-like lymphoma with 11q aberration confirmed by needle biopsy of the liver: A case report

Yang HJ, Wang ZM

9478 Common carotid artery thrombosis and malignant middle cerebral artery infarction following ovarian hyperstimulation syndrome: A case report

Xu YT, Yin QQ, Guo ZR

9484 Postoperative radiotherapy for thymus salivary gland carcinoma: A case report

Deng R, Li NJ, Bai LL, Nie SH, Sun XW, Wang YS

9493 Follicular carcinoma of the thyroid with a single metastatic lesion in the lumbar spine: A case report

Ш

Chen YK, Chen YC, Lin WX, Zheng JH, Liu YY, Zou J, Cai JH, Ji ZQ, Chen LZ, Li ZY, Chen YX

#### **Contents**

#### Thrice Monthly Volume 10 Number 26 September 16, 2022

9502 Guillain-Barré syndrome and hemophagocytic syndrome heralding the diagnosis of diffuse large B cell lymphoma: A case report

Zhou QL, Li ZK, Xu F, Liang XG, Wang XB, Su J, Tang YF

9510 Intravitreous injection of conbercept for bullous retinal detachment: A case report

Xiang XL, Cao YH, Jiang TW, Huang ZR

Supratentorial hemangioblastoma at the anterior skull base: A case report 9518

Xu ST, Cao X, Yin XY, Zhang JY, Nan J, Zhang J

#### **META-ANALYSIS**

Certain sulfonylurea drugs increase serum free fatty acid in diabetic patients: A systematic review and 9524 meta-analysis

Yu M, Feng XY, Yao S, Wang C, Yang P

#### **LETTER TO THE EDITOR**

9536 Glucose substrate in the hydrogen breath test for gut microbiota determination: A recommended noninvasive test

ΙX

Xie QQ, Wang JF, Zhang YF, Xu DH, Zhou B, Li TH, Li ZP

9539 A rare cause of acute abdomen after a Good Friday

Pante L, Brito LG, Franciscatto M, Brambilla E, Soldera J

9542 Obesity is associated with colitis in women but not necessarily causal relationship

Shen W, He LP, Zhou LL

9545 Risk stratification of primary liver cancer

Tan YW

#### Contents

#### Thrice Monthly Volume 10 Number 26 September 16, 2022

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CASE REPORT

## Noncirrhotic portal hypertension due to peripheral T-cell lymphoma, not otherwise specified: A case report

Miao-Miao Wu, Wen-Jun Fu, Jia Wu, Lin-Lin Zhu, Ting Niu, Rong Yang, Jin Yao, Qiang Lu, Xiao-Yang Liao

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#### **Abstract**

#### **BACKGROUND**

Peripheral T-cell lymphoma (PTCL), an aggressive and rare disease that belongs to a heterogeneous group of mature T-cell lymphomas, develops rapidly and has a poor prognosis. Early detection and treatment are essential to improve patient cure and survival rates. Here, we report a rare case of PTCL with clinical presentation of noncirrhotic portal hypertension, which provides a basis for early vigilance of lymphomas in the future.

#### CASE SUMMARY

A 65-year-old Chinese woman was admitted to our hospital because of abdominal distension for 3 months and pitting oedema of both lower limbs for 2 months. Physical examinations and associated auxiliary examinations showed the presence of hepatosplenomegaly, and her hepatic venous pressure gradient was 10 mmHg. Immunohistochemical analysis of the liver biopsy confirmed the diagnosis of PTCL. The patient underwent combination therapy with dexamethasone, VP-16, and chidamide. Unfortunately, after 41 days of chemotherapy, the patient died of multiple organ failure.

#### CONCLUSION

PCTL accompanied by noncirrhotic portal hypertension is rarely reported. This

case report discusses the diagnosis of a patient according to the literature.

**Key Words:** Noncirrhotic portal hypertension; Ascites; Peripheral T-cell lymphoma; Lymphoma; Chidamide; Case report

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Core Tip: Peripheral T-cell lymphoma (PTCL) is an aggressive and rare disease that belongs to a heterogeneous group of mature T-cell lymphomas, and is classified as PTCL, not otherwise specified (PTCL-NOS). It is the most common type and most often involves nodal sites; however, many patients present with extranodal involvement, including the liver, bone marrow, gastrointestinal tract, and skin. The clinical presentations of PTCL are lymphadenopathy syndrome and B symptoms (night sweats, fever, and weight loss). Noncirrhotic portal hypertension, hydrothorax and ascites can also occur in rare cases, and noncirrhotic portal hypertension and ascites are less common as first symptoms. Here, we report a rare case of a patient with PTCL who presented with noncirrhotic portal hypertension.

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#### INTRODUCTION

Peripheral T-cell lymphoma (PTCL) is an aggressive and rare disease that belongs to a heterogeneous group of mature T-cell lymphomas, and is classified as PTCL not otherwise specified (PTCL-NOS). It is the most common type and most often involves nodal sites; however, many patients present with extranodal involvement, including the liver, bone marrow, gastrointestinal tract, and skin[1]. The clinical presentations of PTCL are lymphadenopathy syndrome and B symptoms (night sweats, fever, and weight loss). Noncirrhotic portal hypertension, hydrothorax, and ascites can also occur in rare cases [2,3], and noncirrhotic portal hypertension and ascites are less common as first symptoms. Here, we report a rare case of a patient with PTCL who presented with noncirrhotic portal hypertension.

#### **CASE PRESENTATION**

#### Chief complaints

A 65-year-old Chinese woman was referred to our hospital due to abdominal distension for over 3 months and pitting oedema of both lower limbs for over 2 mo.

#### History of present illness

Approximately 3 mo previously, this patient was admitted to a local hospital due to abdominal distension. Additional symptoms included pitting oedema of both lower limbs, anorexia and melena, and the patient lost 5 kg in two months. The patient had no jaundice, fever, or night sweats. Ultrasonography (US) indicated that she had neither oesophageal nor gastric varices or ulcers. but gastroscopic examination showed external compression of large gastric curvature, the cause of gastric wall compression is unknown, taking into account the left side of lying stomach bend side close to the spleen. Extra-hospital computed tomography (CT) showed hepatosplenomegaly and no other lesions around the stomach. So we think the growth of the spleen is the most likely reason for the external pressure of the greater curvature of the stomach. After 10 days of albumin infusion therapy, the above symptoms were not relieved but rather further aggravated. Therefore, she was referred to a tertiary hospital.

#### History of past illness

She had no history of hepatitis and no significant past medical history.

#### Physical examination

The physical examination revealed that the patient had abdominal varices and an increase in abdominal circumference to 98 cm.



#### Laboratory examinations

Laboratory tests showed that her white blood cell count was  $1.80 \times 10^9$ /L (reference value  $3.5-9.5 \times 10^9$ /L), the neutrophil count was  $1.33 \times 10^{9}$ /L (reference value  $1.8-6.3 \times 10^{9}$ /L), the neutrophil percentage was 73.80% (reference value 40%-75%), haemoglobin was 71 g/L (reference value 115-150 g/L), platelet count was 54 × 10<sup>9</sup>/L (reference value 100-300 × 10<sup>9</sup>/L), albumin was 26.5 g/L (reference value 40.0-55.0 g/L), lactate dehydrogenase (LDH) was 347 IU/L (reference value 120-250 IU/L), and liver and renal function were within normal limits. Her erythrocyte sedimentation rate was 3.0 mm/h (reference value < 38 mm/h). Coagulation function revealed a prothrombin time of 18.2 s (reference value 9.6-12.8 s), and fibrinogen was 0.64 g/L (reference value 2.0-4.0 g/L) (Table 1). Tests for Epstein-Barr virus, tuberculosis, and hepatitis B, A, C, D, and E were all negative.

#### Imaging examinations

Positron emission tomography-CT (PET) indicated hepatosplenomegaly and swelling of the peritoneum, mesentery, descending duodenum, and horizontal segments (Figure 1). Liver ultrasound showed increased liver volume; no cirrhosis-specific nodular changes were observed, and liver stiffness was measured at 19.55 kPa (Figure 2). CT also showed an enlarged liver volume, parenchymal density, and acceptable strengthening, as well as a small, tiny cyst. Two sides of the intrahepatic portal vein showed no enhancement of the low-density strip, showing a "double-track sign". Consideration was given to the possibility of intrahepatic lymphatic stasis. Liver computed tomographic arteriography showed hepatosplenomegaly without portal vein, splenic vein, or mesenteric arteriovenous thrombosis

#### FURTHER DIAGNOSTIC WORK-UP

Relevant laboratory data for ascites were as follows: appearance was yellow and cloudy, nucleated cells were 40 × 106/L, red blood cells were 6700 × 106/L, albumin was 25.3 g/L, glucose was 6.85 mmol/L, LDH was 246 IU/L, serum-ascites LDH gradient was 0.87, adenosine deaminase was 27.8 IU/L, the serum-ascites albumin gradient (SAAG) was 1.3, the ascitic fluid bacterial culture showed no bacterial growth, and the tuberculosis antibody test was negative. The hepatic venous pressure gradient (HVPG) measured by transjugular intrahepatic portosystemic shunt (TIPS) was 10 mmHg.

Ascites immunocytology showed that the lymphocyte population composition was approximately 93.3% nucleated cells, of which approximately 14.5% were weakly positive for CD5. Immunohistochemistry staining showed that the lymphocytes were positive for CD2, CD3, CD7, CD8, CD56, CD57, and T-cell receptor (TCR) αβ but negative for CD11c, CD16, CD4, and TCR γδ. Because of the limitation of the medical technique used, DNA ploidy analysis of the exfoliated cells of the benign and malignant hydrothorax as well as flow cytometry of the ascites were not performed. Immunohistochemical analysis, performed on a bone marrow biopsy and aspiration (Figure 4), showed that cells were positive for CD3, CD7, and CD56 and negative for CD20, CD2, CD5, CD4, CD8, TIA-1, CD30, and CD34, as well as for Epstein-Barr virus-encoded RNAs, using in situ hybridization. Medical examination of ascites and bone marrow revealed a T-cell lymphoma. HVPG was measured by TIPS, liver biopsy was performed, and immunohistochemistry was performed. Macroscopically, 4 strips of grey yellow cordlike tissue, approximately 0.8-1.3 cm in length and 0.1 cm in diameter, were positive for CD3, CD5, CD7, CD8, CD43, CD56, TIA-1, and Ki-67 and negative for CD2, CD20, CD4, CD30, CD34, and TdT (Figure 5). Gene rearrangements are seen in the TCR γ low amplification peak (Table 2).

#### **FINAL DIAGNOSIS**

Eventually, after twenty days, the patient was diagnosed with an aggressive T-cell lymphoma (stage IV), which was categorised as a PTCL-NOS.

#### TREATMENT

The patient was diagnosed with T-cell lymphoma and was given 5 mg dexamethasone once a day. After four days of hormone therapy, the patient's oedema of both lower extremities was alleviated, but the reduction was not obvious; therefore, 25 mg etoposide once a week was added. After two days, the oedema of both lower limbs was significantly improved; however, ascitic changes were unremarkable. After 12 days, the patient was put on chidamide chemotherapy at the recommended starting dose of 20 mg, 1 to 3 times per week, 20 to 50 mg each time; the patient took the medication for 22 days with a cumulative dose of 110 mg, and the oedema of both lower limbs disappeared completely; however, ascites remained unchanged.

9419

Table 1 Main laboratory test results						
Laboratory tests	Result	Reference value				
White blood cell count	1.80 × 10 <sup>9</sup> /L	3.5-9.5 × 10 <sup>9</sup> /L				
Neutrophil count	$1.33\times10^9/L$	$1.8-6.3 \times 10^9/L$				
Neutrophil percentage	0.738	40-75%				
Haemoglobin	71 g/L	115-150 g/L				
Platelet count	$54 \times 10^9 / L$	$100-300 \times 10^9/L$				
Albumin	26.5 g/L	40.0-55.0 g/L				
Lactate dehydrogenase	347 IU/L	120-250 IU/L				
Erythrocyte sedimentation rate	3.0 mm/h	< 38 mm/h				
Coagulation function revealed a prothrombin time of	18.2 s	9.6-12.8 s				
Fibrinogen	0.64 g/L	2.0-4.0 g/L				

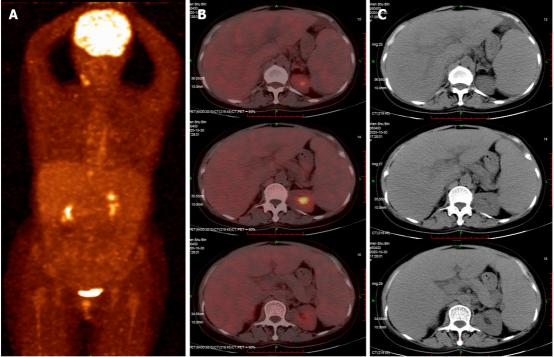
Table 2 Immunohistochemi	stry of pleural fluid, ascites	s, liver, and bone marrow		
Parameter	Marrow	Ascites	Hydrothorax	Liver
CD2	++	+	+	-
CD3	++	++	+	+
CD4	-	-	-	-
CD5		+	+	+
CD7	++	++	++	+
CD8	+	+	++	+
CD11c	-	-		
CD16	-	+	-	
CD30				-
CD34				-
CD38		-	++	
CD43				+
CD45		+		
CD56	++	++	++	+
CD57	+	+p	++	
ΤCRαβ	+	+		
ΤCRγδ	-	-		
TIA-1				-
TDT				-
Ki-67				+

<sup>&</sup>quot;+" indicates positive, the number indicates positive degree; "-" indicates negative; blank indicates that the examination was not performed.

#### **OUTCOME AND FOLLOW-UP**

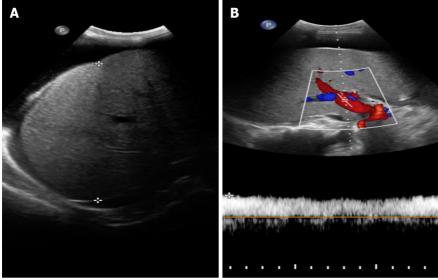
The patient's international prognostic index was 5, which meant that she was in the high-risk group with a low 5-year survival rate[4]. Chidamide has a slower onset of action, and over the course of her treatment, her ascites did not improve. Unfortunately, after 41 d of chemotherapy, the patient died of multiple organ failure.





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Figure 1 Positron emission tomography. A: whole body scan; B and C: Scan showing the liver (SUVmax 2.28, mean 1.90), The liver was diffusely enlarged, increased spleen volume was observed, and there was a small amount of abdominal effusion.

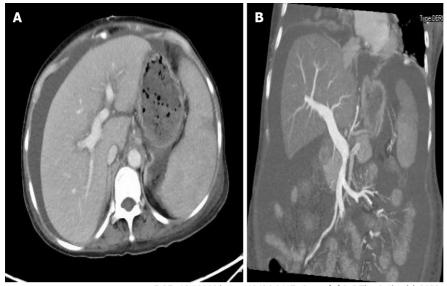


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Figure 2 Liver ultrasound. A: The liver capsule was less smooth, and the right liver had a maximum oblique transaxial distance of 15 cm and increased parenchymal echogenicity. The liver parenchyma was slightly thickened and heterogeneous without a definite space-occupying lesion; B: The diameter of the extrahepatic portal vein was approximately 12 mm, and the blood flow was unidirectional to the liver at a flow rate of 29.3 cm/s. The cava caliber and lumen appeared normal, as did blood flow in the hepatic vein, superior mesenteric vein, splenic vein, and inferior vena cava.

#### **DISCUSSION**

PTCL is an aggressive and rare disease that belongs to a heterogeneous group of mature T-cell lymphomas that constitute less than 15% of all non-Hodgkin lymphomas in adults [5,6]. The most common symptoms included lymphadenopathy and B symptoms (night sweats, fever, and weight loss). The initial findings in the patient were portal hypertension, ascites, splenomegaly, and routine biochemistry of ascites showing no infection. SAAG was 1.3, and HVPG was 10 mmHg. Ultrasound and CT showed hepatosplenomegaly and liver cirrhosis. CTA showed that there was no obstruction or



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Figure 3 Liver computed tomographic arteriography. A: Spleen growth: spleen enhancement was less uniform, and patchy slightly hypointense areas were seen, not excluding infarcts or intrahepatic lymphatic stasis; B: The main portal vein and splenic vein were slightly thickened, the diameter of the main portal vein was approximately 1.5 cm, and there was no thrombus or collateral circulation opening, A and B: The vessel was smooth and unobstructed.

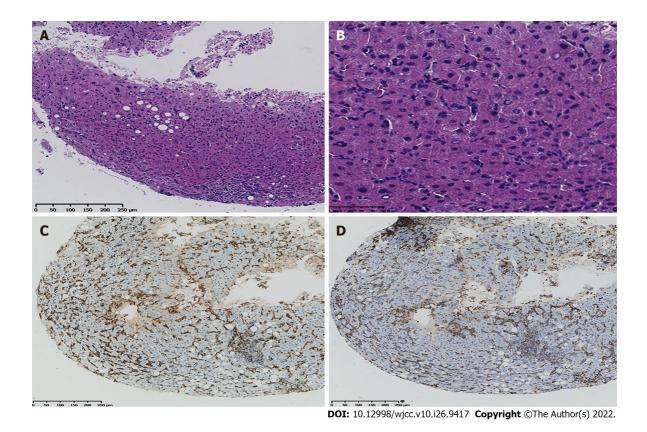


Figure 4 Pathology and immunochemistry were performed on four pieces of liver tissue. A: Histopathological examination by haematoxylin and eosin staining (100 x); B: Histopathological examination by haematoxylin and eosin staining (400x) showed few hepatocytes that were watery and lipomatous and multiple small focal lymphoid cell infiltrates; C: Immunochemical staining shows CD3 positivity; D: Immunochemical staining shows CD7 positivity. H&E, haematoxylin

compression of blood vessels before and after the trunk. Therefore, we excluded prehepatic and posthepatic portal hypertension and focused on intrahepatic portal hypertension[7], and the patient's intrahepatic lymphatic stasis confirmed this speculation[8]. Although all examinations of our patients showed cirrhosis, the patient's liver was enlarged, so we had to doubt the accuracy of the examination. To our knowledge, the gold standard for cirrhosis is liver biopsy, which can be performed for diagnostic

and eosin.

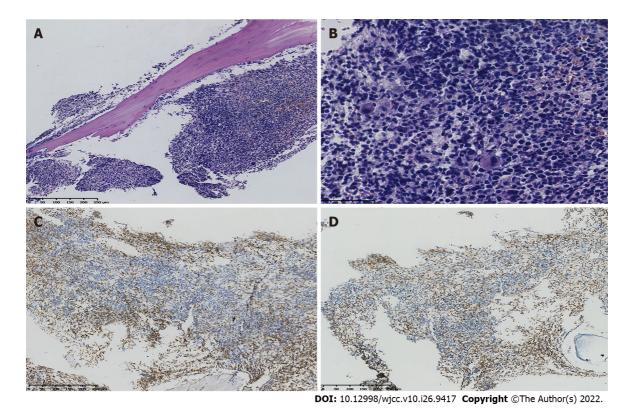


Figure 5 Pathology and immunochemistry of bone marrow. A: Histopathological examination by haematoxylin and eosin staining (100 x); B: Histopathological examination by haematoxylin and eosin staining (400x) shows nucleated cells actively proliferating and replacing most of the adipose tissue, with more red blood cells than granulocytes and 2-4 megakaryocytes/hpf. The pathology showed that the bone marrow was nucleated and cell proliferation was active; C: Immunochemical staining shows CD3 positivity; D: Immunochemical staining shows CD7 positivity. H&E, haematoxylin and eosin; HPF, high power field.

purposes when the diagnosis is uncertain. Before they are properly diagnosed, most patients are misdiagnosed as having hepatic cirrhosis, potentially delaying treatment. Consequently, the patient was subjected to a liver biopsy which confirmed the absence of cirrhosis; thus, this presentation was either PTCL or NOS (Figure 6).

Portal hypertension is a rare manifestation in lymphomas. By searching cases of PTCL-related portal hypertension and ascites (Table 3), we found that only one report[9] described a T-cell lymphoma patient presenting with portal hypertension and oesophageal and gastric varices but no ascites and whose diagnosis was confirmed by splenectomy. Four cases [10-13] mentioned ascites, but none reported whether there was portal hypertension and only reported diagnosis by ascites flow cytometry. The mechanism of portal hypertension is not mentioned in the above cases. We speculate that the reason for the noncirrhotic portal hypertension in this patient was intrahepatic portal hypertension caused by the obstruction of a portal venous return due to intrahepatic lymph stasis. At the same time, visceral hyperdynamic circulation is one of the causes of increased portal blood flow and thus increased portal venous system pressure [14-16]. This case may be due to increased liver blood flow caused by tumours resulting in visceral hyperdynamic circulation leading to liver enlargement and portal hypertension. These two mechanisms together led to the development of noncirrhotic portal hypertension in this patient.

In the relevant literature, most patients are diagnosed based on ascites flow patterns, but in this case, due to insufficient sampling, the ascites flow pattern could not be successfully made. TIPS was used to perform portal pressure measurement and liver biopsy in this patient, and the diagnosis was finally made based on the biopsy results. TIPS is an interventional radiotherapy technique developed in the past 20 years. It uses the internal jugular vein as the puncture entrance, inserts the catheter through the superior vena cava, right atrium, and inferior vena cava, and inserts the hepatic vein into the hepatic vein under the guidance of an X-ray. Establishing an artificial shunt channel between them can not only measure the portal venous pressure to achieve the purpose of diagnosis but also reduce portal hypertension and achieve the purpose of treatment [17-19]. TIPS has been widely used for the diagnosis and treatment of portal hypertension[20]. TIPS is considered to be a successful and efficacious procedure with a 90% success rate [21,22]. Although there are unavoidable risks, recent studies have shown a high efficacy of TIPS compared to other treatments and presented an acceptable complication rate[23,24]. Research has shown that TIPS placement can be used for noncirrhotic portal hypertension [25]. At the same time, TIPS can also be used for liver biopsy to obtain a pathological diagnosis. When the patient was diagnosed with portal hypertension by TIPS, a liver biopsy was also performed, which

Table 3 A summary of demographic, radiographic, and clinical information from a review of five previously published cases of T-cell lymphomas with ascites and/or portal hypertension manifestations

Ref.	Age, Gender	Course of disease	Clinical Symptoms	Supplementary Examination	Biopsy Source	Immunohistochemistry	Diagnosis	Invasion of other parts	Treatment	Prognosis
Ameri[13]	61, F	2 <sup>+</sup> W	Abdominal discomfort	Ascites, hepatospleno- megaly	Ascites	CD4(+), CD2(+), CD5(+), CD3(+), CD7(-), CD16(-), CD56(-), CD57(-), TdT(-)	PTCL, NOS	Bone marrow	No treatment	NA
Yamamoto [10]	72, W	3 <sup>+</sup> W	Abdominal discomfort	Hydrothorax and ascites	Ascites	CD2 (+), CD3(+) (+),CD45(+), CD4 (-), CD8 (-)	PTCL	Thorax and abdomen	Cyclophosphamide, mitoxantrone, vincristine, etoposide, bleomycin, and prednisolone	Died of multiple organ failure
Izban[12]	76, F		Abdominal tenderness	Ascites, splenomegaly	Ascites	CD2(+), CD3(+), CD5(+), CD7(+), CD45(+), CD4(-), CD8(-)	PTCL	Bone marrow, liver	CHOP chemotherapy	Recurrence after chemotherapy
VakarLópez [11]	49, W	3 <sup>+</sup> M	Abdominal tenderness	Ascites	Ascites	CD3(+)	PTCL, NOS		No treatment	NA
Lindor[9]	65, F	2 <sup>+</sup> Y	Pectoralgia, esophageal and gastric variceal bleeding (EGVB)	Splenomegaly, EGVB	spleen	NA	Diffuse mixe lymphoma	ed-type T-cell	Splenectomy	Bone marrow infiltration occurred 1 + year after the operation

PTCL: Peripheral T-cell lymphoma; NOS: Not otherwise specified.

was the key to the final diagnosis of the patient.

Therefore, when noncirrhotic portal hypertension is the main manifestation, all examinations suggest liver cirrhosis, but imaging does not conform to the characteristics of liver cirrhosis, such as liver enlargement. TIPS examination can be considered, which can not only measure portal pressure but also perform a biopsy to achieve the purpose of diagnosis, and the examination risk is relatively low. This study is helpful to reduce the missed diagnosis rate of lymphoma. PTCL-NOS has a poor prognosis, and the commonly used CHOP regimen is not effective[26-29]. Chidamide also takes at least 4 weeks to take effect. Early diagnosis can lead to more treatment opportunities and increase the prognosis of patients.

#### **CONCLUSION**

In conclusion, we describe a PTCL case presenting with ascites and noncirrhotic portal hypertension. Cases of noncirrhotic portal hypertension in PTCL are rare. When the clinical signs and auxiliary examinations suggest liver cirrhosis, as long as there is noncompliance with liver cirrhosis (hepatosplenomegaly), we should be alert to the possibility of other causes, such as lymphoma, reducing missed diagnosis of lymphoma.

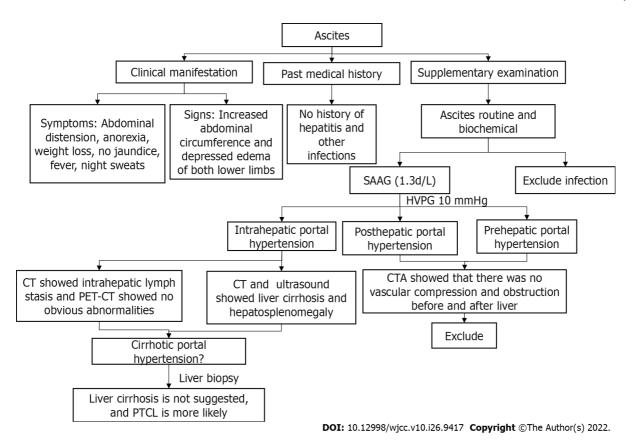


Figure 6 Diagnosis flow charts. PTCL: Peripheral T-cell lymphoma; CT: Computed tomography; CTA: Computed tomography angiography; SAAG: Serumascites albumin gradient; PET-CT: Positron emission tomography-CT.

#### **FOOTNOTES**

Author contributions: Liao XY designed and performed the research; Wu MM analysed the data and wrote the paper; Wu J, Niu T, and Fu WJ cared for the patient, designed and analysed the research, and helped with writing the paper; Zhu LL, and Yang R participated in revising the manuscript; Lu Q carried out the ultrasound; Yao J performed imaging studies; all authors have read and approved the final manuscript.

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9425

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9426

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9427



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