

Isolated peritoneal lymphomatosis defined as post-transplant lymphoproliferative disorder after a liver transplant: A case report

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Author contributions: Kim HB were the major contributors in writing the manuscript; Na YS and Choi WY advised the manuscript; Park SG and Lee HJ were involved in drafting, writing and editing the manuscript; Lee HJ reviewed the manuscript as corresponding author; All authors read and approved the final manuscript.

Supported by the National Research Foundation of Korea, No. NRF-2017R1C1B5076793.

Informed consent statement: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict-of-interest statement: The authors declare no conflict-of-interest.

CARE Checklist (2016) statement: The manuscript was prepared and revised according to the CARE Checklist (2016).

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Abstract

BACKGROUND

Post-transplant lymphoproliferative disorder (PTLD) is a fatal complication of solid organ transplantation or allogenic hematopoietic stem cell transplantation that is associated with immunosuppressive therapy. Potential manifestations are diverse, ranging from reactive lymphoid hyperplasia to high-grade lymphoma. PTLD is usually of B-cell origin and associated with Epstein-Barr virus (EBV) infection. Herein, we describe a case of PTLD involving the peritoneal omentum. There has been only case of PTLD as a diffuse large B-cell lymphoma (DLBCL) in the peritoneum.

CASE SUMMARY

The patient was a 62-year-old man who had been receiving immunosuppressive therapy with tacrolimus since undergoing a liver transplant 15 years prior. He reported that he had experienced abdominal discomfort and anorexia 1 month prior to the current admission. Abdominal pelvic computed tomography (CT) revealed peritoneal and omental mass-like lesions without bowel obstruction. Ultrasonography-guided biopsy was performed, and he was histologically

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Manuscript source: Unsolicited manuscript

Received: October 7, 2019

Peer-review started: October 7, 2019

First decision: October 23, 2019

Revised: October 31, 2019

Accepted: November 15, 2019

Article in press: November 15, 2019

Published online: December 26, 2019

P-Reviewer: Nacif L, Shrestha B, Sun WB

S-Editor: Ma YJ

L-Editor: A

E-Editor: Liu JH



diagnosed with EBV-negative DLBCL. Positron emission tomography (PET)-CT depicted peritoneum and omentum involvement only, without any lymphadenopathy or organ masses, including in the gastrointestinal tract. Six cycles of chemotherapy with a “R-CHOP” regimen (rituximab-cyclophosphamide, doxorubicin, vincristine, prednisolone) were administered, and PET-CT performed thereafter indicated complete remission.

CONCLUSION

This is the first report of isolated peritoneal lymphomatosis defined as PTLD in a liver transplant recipient.

Key words: Case report; Diffuse large B-cell lymphoma; Epstein-Barr virus infection; Posttransplant lymphoproliferative disorder; R-CHOP; Isolated peritoneal lymphomatosis

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Core tip: Post-transplant lymphoproliferative disorder (PTLD) is a fatal complication of solid organ transplantation or allogeneic hematopoietic stem cell transplantation that is associated with immunosuppressive therapy. Herein we present the first report of isolated peritoneal lymphomatosis defined as PTLD in a liver transplant recipient.

Citation: Kim HB, Hong R, Na YS, Choi WY, Park SG, Lee HJ. Isolated peritoneal lymphomatosis defined as post-transplant lymphoproliferative disorder after a liver transplant: A case report. *World J Clin Cases* 2019; 7(24): 4299-4306

URL: <https://www.wjnet.com/2307-8960/full/v7/i24/4299.htm>

DOI: <https://dx.doi.org/10.12998/wjcc.v7.i24.4299>

INTRODUCTION

Post-transplant lymphoproliferative disorder (PTLD) is a fatal disease that occurs in patients who have undergone solid organ transplantation and allogeneic hematopoietic stem cell transplantation. Its incidence varies depending on the organ transplanted, and after liver transplantation the incidence of PTLD is approximately 1.0%–5.5%^[1,2]. Epstein-Barr virus (EBV) infection is known to be the most common etiology. It is typically classified into four histologic types; early lesion, polymorphic, monomorphic, and Hodgkin-like, and it can also be categorized as early-onset (< 2 years) or late-onset (≥ 2 years)^[3-5]. PTLD in the form of extranodal lymphoma is slightly more common than other forms, and the gastrointestinal tract, transplanted organs, and central nervous system are the most frequently affected sites^[2,6-9]. To date, no cases of peritoneal lymphomatosis have been reported.

Herein, we describe the case of a patient with diffuse large B-cell lymphoma (DLBCL) constituting late-onset monomorphic PTLD, which developed 15 years after a liver transplant. The case involved isolated peritoneal lymphomatosis, which has never been reported in the context of PTLD, and complete remission was achieved *via* chemotherapy.

CASE PRESENTATION

Chief complaints

A 62-year-old man was admitted due to reduced appetite and abdominal distension, which he had reportedly been experiencing for the last month.

History

The patient had been followed up regularly since undergoing a liver transplant 15 years prior due to fulminant hepatic failure associated with hepatitis B, and he had been taking tacrolimus 2 mg regularly since that transplant. He had been taking linagliptin and metformin for the past 10 years due to diabetes mellitus. Abdominopelvic computed tomography (CT) had been conducted 6 months prior to the current presentation as part of a periodic check-up, and it had not depicted any abnormal findings. He had developed abdominal discomfort 1 month prior to the

current admission, and 2 weeks after its initial onset he developed abdominal distension that was so severe that he could not eat. He then visited the hospital for testing.

Physical examination upon admission

There was no tenderness upon abdominal palpation but distension was severe, with fluid wave and shifting dullness indicating ascites.

Laboratory examinations

Complete blood count results were as follows, with normal ranges in parentheses: white blood cells $2.1 \times 10^3/\mu\text{L}$ (4.0–10.0 $10^3/\mu\text{L}$), hemoglobin 13.8 g/dL (12–16 g/dL), platelets $318 \times 10^3/\mu\text{L}$ (150–400 $10^3/\mu\text{L}$). Blood biochemistry results were total bilirubin 0.6 mg/dL (0.2–1.1 mg/dL), aspartate aminotransferase 21 U/L (5–40 U/L), alanine aminotransferase 9 U/L (5–40 U/L), albumin 3.7 g/dL (3.5–5.2 g/dL), blood urea nitrogen 14.4 mg/dL (8.0–20.0 mg/dL), creatinine 0.9 mg/dL (0.5–1.3 mg/dL), C-reactive protein 10.8 mg/dL (0.0–0.5 mg/dL). Among the tumor markers tested for, carcinoembryonic antigen was normal (1.3 ng/mL; normal range 0.0–5.0 ng/mL) but lactate dehydrogenase was elevated (746 U/L; normal range 200–450 U/L).

Imaging examinations

Abdominopelvic CT depicted diffuse peritoneal thickening, omental masses and nodules, and ascites with omental fat infiltration, but no mass-like lesions or bowel obstruction were evident in the gastrointestinal tract (Figure 1). A primary peritoneal disease such as tuberculous peritonitis or malignant mesothelioma was suspected. Positron emission tomography (PET)-CT was performed, and it did not depict abnormal hypermetabolism in solid organs, lymph nodes, or digestive organs but it did reveal diffuse hypermetabolism in the peritoneum and omentum (Figure 2).

FINAL DIAGNOSIS

Histological examination was performed on the omental mass using a percutaneous ultrasonography guided core needle, and the mass was definitively diagnosed as DLBCL that was positive for CD20, CD45, and B-cell lymphoma 6, and negative for CD3 and cytokeratin (Figure 3). Other tissue markers were negative for EBV and human herpesvirus 8 (HHV-8). Serum EBV PCR was negative as was serum EBV viral capsid IgM testing, indicating that manifest EBV infection was not present. Serum EBV viral capsid IgG testing was positive. Hepatitis B antigen testing was negative, hepatitis B antibody testing was positive, and hepatitis C antibody testing was negative. Based on these results, the patient was diagnosed with DLBCL as an EBV-negative monomorphic PTLD that had developed 15 years after a liver transplant. The condition was further classified as an isolated peritoneal stage IIE DLBCL with lymph node infiltration but no bone marrow or other solid organ infiltration. The International Prognostic Index score was in the intermediate-low-risk group, as the patient was 62 years of age with elevated lactate dehydrogenase.

TREATMENT

The dosage of tacrolimus, an immunosuppressor, should be reduced in patients with PTLD, but he did not want to reduce the dose of the tacrolimus; the tacrolimus level was maintained at a slightly low level of 4. Two cycles of an “RCHOP” (rituximab-cyclophosphamide, doxorubicin, vincristine, prednisolone) regimen, the standard treatment for DLBCL, were administered without a dosage reduction. While on chemotherapy diabetes was not controlled, so response evaluation was performed *via* CT. CT depicted marked peritoneal mass reduction, indicating a partial response, so 6 cycles were administered without dosage reduction. Subsequent CT scanning did not depict any signs of omental mass or ascites, but there was still mild haziness in the omental fat (Figure 1). PET-CT following an aggressive diabetes management regimen indicated complete remission, with loss of diffuse hypermetabolism lesion in the peritoneum (Figure 2).

OUTCOME AND FOLLOW-UP

Although PET-CT suggested complete remission, CT was performed 2 mo later to monitor the previously observed mild haziness on omentum fat. The haziness had

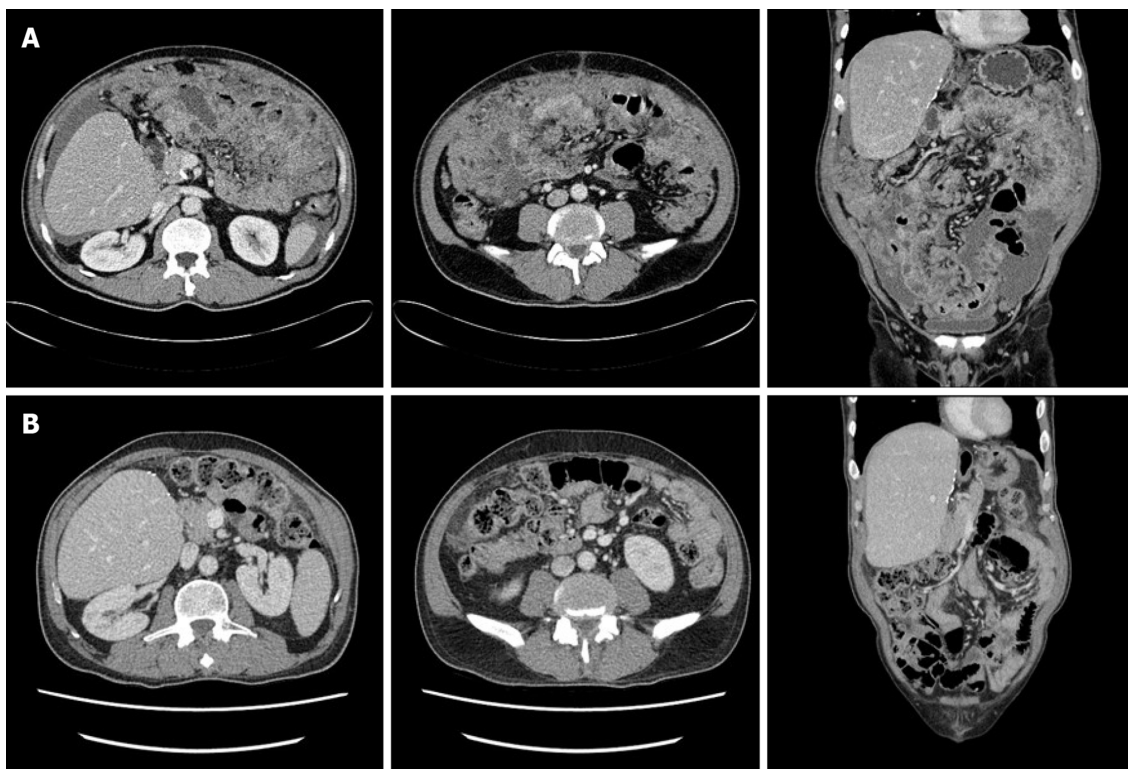


Figure 1 Abdominopelvic computed tomography. A: Computed tomography (CT) depicting a large volume of ascites and diffuse peritoneal infiltrative lesions at the mesentery and omentum, but no mass-like lesions in the gastrointestinal tract and no bowel obstruction; B: Post-chemotherapy CT depicting no signs of omental mass or ascites, but mild haziness in the omental fat.

reduced, suggesting that the omentum was undergoing a healing process. The patient currently remains under observation.

DISCUSSION

PTLD is defined as a lymphoma that develops after solid organ or hematopoietic stem cell transplantation. Since some researchers reported five cases of PTLD as a severe complication of transplantation in 1966, the incidence of PTLD has risen in conjunction with an exponential increase in the number of transplants performed. Although the incidence varies depending on the organ transplanted and the use of immunosuppressors, the incidence of PTLD in cases of liver transplantation is approximately 1.0%–5.5%^[1,2]. Of the four pathological types, monomorphic PTLD exhibits the typical characteristics of a malignant lymphoma. DLBCL is the most common, followed by Burkitt lymphoma and aggressive T-cell lymphoma^[10].

Approximately 50%–70% of PTLD cases are early-onset, more than 80% of these early onset cases are of B-cell origin, and more than 90% are EBV-positive^[4,11–13]. Conversely, EBV-negativity is more common in cases of late-onset PTLD^[14]. EBV-induced pathogenesis of PTLD is relatively well known. With immunosurveillance by T lymphocytes diminished *via* immunosuppressors, which are used to prevent rejection responses in transplant recipients, primary infection or reactivation of EBV may thwart regulation of the proliferation of transformed B-cells, ultimately causing PTLD^[15,16].

The mechanisms involved in EBV-negative PTLD remain unclear. The “hit and run” hypothesis suggests that EBV infection occurs but is then lost after causing PTLD^[17,18]. Some reports suggest that other viruses such as HHV-8, hepatitis B virus, hepatitis C virus, and cytomegalovirus may be causative, and some reports note that PTLD can have similar characteristics to p53 mutation lymphoma^[19–23]. The manifestation of PTLD varies widely, from asymptomatic to fulminant onset. Extranodal lymphoma is relatively common, and the gastrointestinal tract (20%–30%), transplanted organ (10%–15%), and central nervous system (5%–20%) are commonly affected^[2,6–9]. To date however, there have been no reports of primary peritoneal onset as was observed in the present case.

PTLD has a poorer prognosis than malignant lymphoma, and the 5-year survival

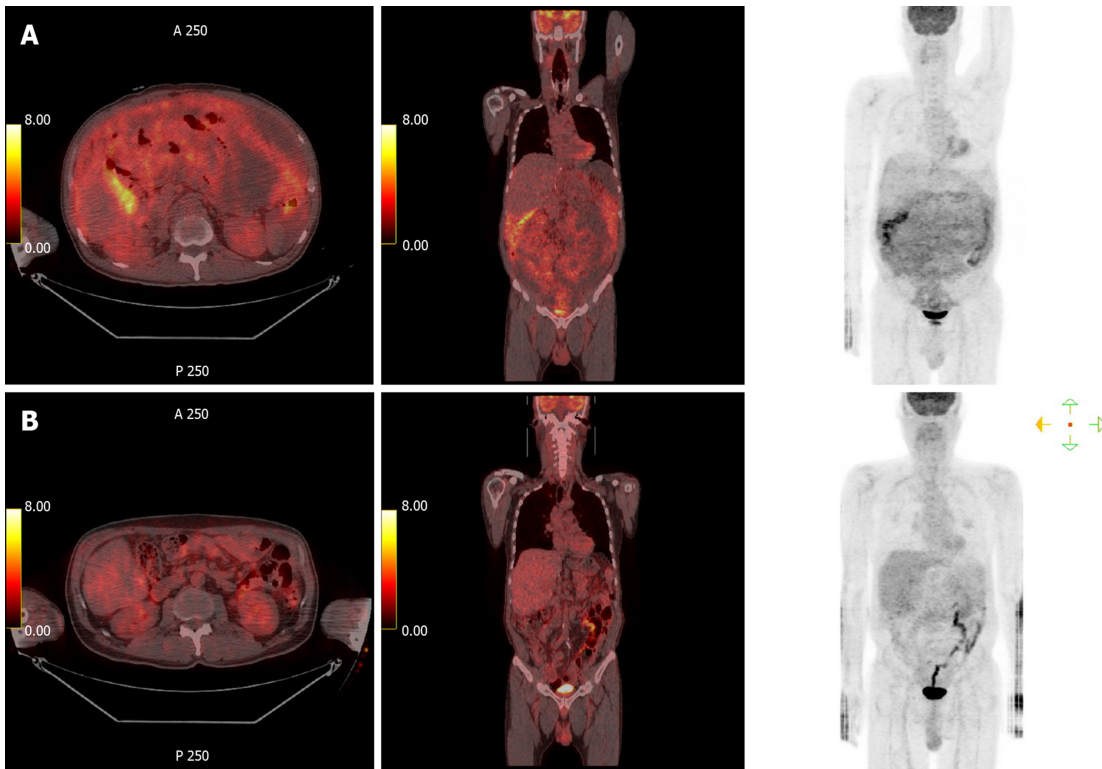


Figure 2 Positron emission tomography-computed tomography. A: Positron emission tomography (PET)-computed tomography (CT) did not depict abnormal hypermetabolism in solid organs, lymph nodes, or digestive organs, but it did depict diffuse hypermetabolism in the peritoneum and omentum; B: Post-chemotherapy PET-CT depicted loss of diffuse hypermetabolic lesion in the peritoneum.

rate of DLBCL as a PTLD is approximately 40%–60%^[2,9,24]. It is not clear whether the prognosis of malignant lymphoma that develops as PTLD is poor or whether transplant recipients' underlying disease and reduced organ function and infection due to immunosuppressors are the main problems, but the International Prognostic Index score is evidently an important prognostic factor^[2,25,26].

For PTLD treatment, lowering the dose of immunosuppressors is suggested in most cases if it has not manifested as an EBV-related aggressive lymphoma, but in practice, this is difficult. It has been reported that rituximab therapy alone can be effective in DLBCL, but the R-CHOP regimen is still considered the standard treatment; thus, further research is warranted^[27,28].

It is known that malignant lymphoma can develop in any part of the body, but malignant lymphomas that involve the omentum or peritoneum are very rare^[29]. Peritoneal lymphomatosis progresses in a manner similar to peritoneal cancer metastasis, where peritoneal thickening with malignant ascites and diffuse infiltration of omental fat-known as omental cake-are observed and the patient complains of severe abdominal discomfort and distension. Usually high-grade malignant lymphoma severely infiltrates the gastrointestinal tract or abdominal lymph node and the peritoneum, but isolated peritoneal lymphomatosis that is restricted to the peritoneum and omentum without involving other gastrointestinal organs is very rare, with no such cases reported as a PTLD to date^[30-34]. Thus, there are no data available on the likely prognosis of the present patient.

Some potentially relevant observations pertaining to therapeutic effects and prognoses of primary effusion lymphoma, a body cavity-based lymphoma, may warrant consideration. Primary effusion lymphoma is a rare but officially acknowledged DLBCL in the WHO classification system that specifically occurs in the serosal surface of the pleura or pericardium in the body cavity^[35]. HHV-8, which occurs as a result of human immunodeficiency virus infection, is known to be the major cause of primary effusion lymphoma but it commonly affects patients receiving immunosuppressant therapy after a transplant^[36]. Given the growing number of reports of HHV-8-negative primary effusion lymphoma, some studies suggest that it should be differentiated from HHV-8-positive lymphoma, but relevant research data are scarce^[37]. However, in light of the fact that it occurs in immunosuppressed patients and in the body cavity, the possibility of peritoneal expression of primary effusion lymphoma can be considered.

Peritoneal lymphomatosis is treated with chemotherapy, and unlike peritoneal

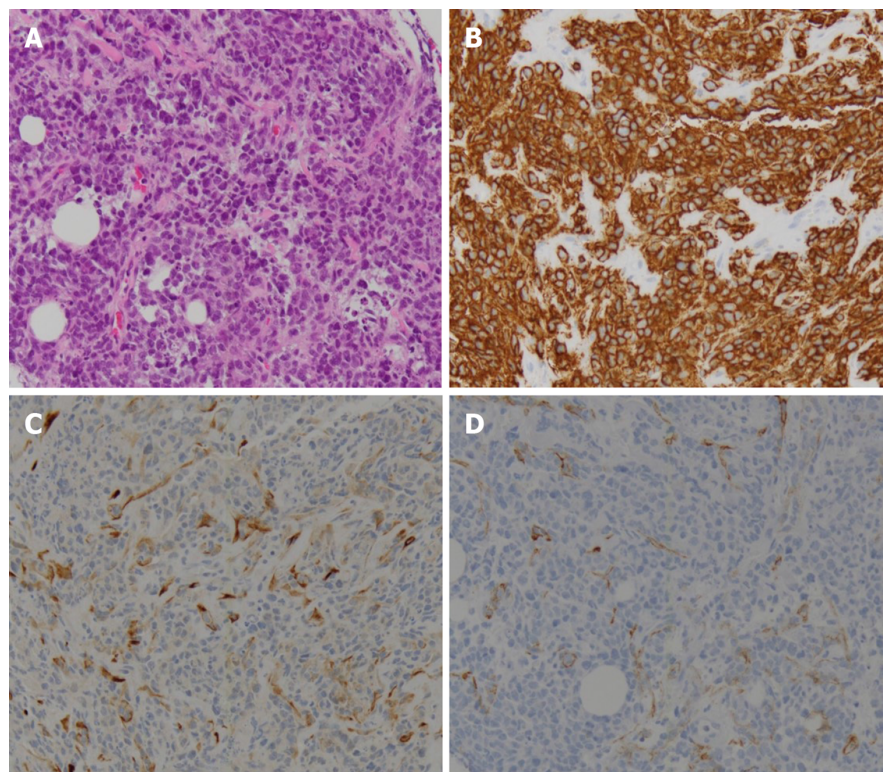


Figure 3 Pathologic findings. Highly pleomorphic, atypical, small, round, blue cells were observed infiltrating the fibrocollagenous tissue (A). On immunohistochemical testing, these cells were positive for CD20 (B) and LCA, but they were negative for the mesothelial cell marker D2-40 (C) and the epithelial cell marker cytokeratin (D).

carcinomatosis, it is curable. Although therapeutic efficacy is predicted to be low due to problems such as reduced peritoneal infiltration by anticancer agents, relevant data are lacking. The present patient is in stage II and classified as low-to-intermediate-risk based on the International Prognostic Index, and PTLD has a relatively low cure rate. Thus, despite the fact that he has currently achieved complete remission, close follow-up will be maintained, given the poor prognosis of primary pleural effusion lymphoma and the possibility of relapse.

CONCLUSION

This is a valuable report as it presents an extremely rare case of isolated peritoneal lymphomatosis after liver transplantation. Moreover, it is the first report of isolated peritoneal lymphomatosis defined as PTLD in a liver transplant recipient.

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