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*Case report*

# High-grade B-cell lymphoma, not otherwise specified, presenting as primary peritoneal lymphomatosis and successfully treated with dose-adjusted EPOCH-R

Akihito Fujimi,[1)] Yasuhiro Nagamachi,[1)] Naofumi Yamauchi,[1)] Naoki Onoyama,[2)] Naotaka Hayasaka,[2)] Teppei Matsuno,[2)] Kazuhiko Koike,[2)] Yoshiro Goto,[2)] Kohji Ihara,[2)] Junji Kato,[2)] Takuji Nishisato,[2)] Hiroshi Kawase,[3)] Tomoyuki Yano,[3)] Takayuki Kanaseki,[4)] Shintaro Sugita,[5)] Masayoshi Kobune[6)]

Peritoneal lymphomatosis (PL) is a rare lymphoma-associated condition defined as the dissemination of lymphoma cells in the peritoneum. An 82-year-old man presented with abdominal pain, heartburn, and high fever. Radiological findings, including positron emission tomography-computed tomography (PET-CT), and gastrointestinal fiberscopy, showed diffuse thickening of the peritoneum, omentum, and mesentery; however, no lymphadenopathy, hepatosplenomegaly, or gastrointestinal lesions were observed. Under suspicion of peritonitis carcinomatosa of unknown origin, exploratory laparoscopy was performed that revealed multiple white nodules and masses on the surfaces of the peritoneum, mesentery, and intestinal serosa. The histopathological and cytogenetic findings of the peritoneum revealed high-grade B-cell lymphoma, not otherwise specified, and a gain of *MYC* by fluorescence *in-situ* hybridization. The patient was treated with two cycles of R-CHOP therapy, followed by six cycles of dose-adjusted EPOCH-R therapy, and a complete metabolic response was confirmed by PET-CT. Since there are no specific radiological findings to confirm the diagnosis of PL, a histopathological diagnosis is usually required. Most PL exhibit an aggressive lymphoma phenotype and can be cured by appropriate chemotherapy. Therefore, early diagnosis and treatment are desirable.

**Keywords:** peritoneal lymphomatosis, high-grade B-cell lymphoma, MYC, DA-EPOCH-R

## INTRODUCTION

Peritoneal lymphomatosis (PL) refers to the dissemination of lymphoma cells in the peritoneum.[1] Although PL may rarely be observed in the advanced stage of lymphoma, development of PL at initial diagnosis is very rare, reportedly occurring in 0.75% of all lymphoma cases.[2] As radiological findings of PL resemble those of peritonitis carcinomatosa (PC), diagnosis of PL without histopathological findings is difficult.[3–5] The prognosis of PC is generally poor, whereas PL can be cured by appropriate chemotherapy. Thus, early and accurate diagnosis of PL is crucial.

High-grade B-cell lymphoma (HGBL), not otherwise specified (NOS), is a rare aggressive mature B-cell lymphoma accounting for 1–2% of non-Hodgkin's lymphoma.[6] This diagnostic category was first introduced in the 2016 revised fourth edition of the World Health Organization (WHO) classification, and retained in the 2022 fifth edition of the WHO classification.[7,8] HGBL, NOS, lymphoma cells have Burkitt-like or blastoid morphology and do not carry concurrent *MYC* and *BCL2* and/or *BCL6* rearrangements.[7–9] Single *MYC* rearrangements are observed in 8–45% of HGBL, NOS, cases, whereas *MYC* amplification is observed in 11–32% of cases.[6,9,10]

Here, we present a PL case, in which the histopathological diagnosis of HGBL, NOS, accompanied by *MYC* alteration was determined from a laparoscopic biopsy specimen. Subsequent intensive chemotherapy induced complete remis-

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[1)]Department of Hematology, Sapporo Kiyota Hospital, Sapporo, Japan, [2)]Department of Internal Medicine, Sapporo Kiyota Hospital, Sapporo, Japan, [3)]Department of Surgery, Sapporo Kiyota Hospital, Sapporo, Japan, [4)]Department of Pathology, Sapporo Medical University, Sapporo, Japan, [5)]Department of Surgical Pathology, Sapporo Medical University, Sapporo, Japan, [6)]Department of Hematology, Sapporo Medical University, Sapporo, Japan
**Corresponding author:** Akihito Fujimi, Department of Hematology, Sapporo Kiyota Hospital, 1-1-1-1 Shin-ei, Kiyota Ward, Sapporo City 004-0831, Japan. E-mail: akihito.fujimi@gmail.com

sion of the disease.

## CASE PRESENTATION

An 82-year-old man presented with a one-month history of abdominal distension and heartburn. The patient had a medical history of distal gastrectomy for a gastric ulcer at the age of 35 years and had been treated for hypertension, dyslipidemia, hyperuricemia, and post-cerebral infarction without paresis. Esophagogastroduodenoscopy showed hiatal herniation and gastritis; therefore, a proton pump inhibitor was prescribed. However, high fever and abdominal pain emerged after 2 weeks, leading to hospitalization. Laboratory data on admission were as follows: normal complete blood count, lactate dehydrogenase 333 U/L, serum creatinine 2.22 mg/dL, soluble interleukin-2 receptor 6,240 U/mL (reference range 157–474 U/mL), and CA125 435.5 U/mL (reference range <35 U/mL). Positron emission tomography-computed tomography (PET-CT) showed diffuse thickening of the peritoneum, omentum, and mesentery, with a maximum standardized uptake value (SUVmax) of 9.8 for $^{18}$F-fluorodeoxyglucose (FDG) accumulation (Figure 1a–d). A small amount of ascites was also observed; however, pleural effusion, lymphadenopathy, hepatosplenomegaly, or solid organ lesions were not detected (Figure 1a–d). Mild FDG accumulation in the mediastinal lymph nodes (SUVmax = 5.1) was also detected, suggesting reactive lymphadenopathy (Figure 1a). Total colonoscopy, including that of the terminal ileum, revealed no abnormalities. Approximately 2 weeks after admission, exploratory laparoscopy was performed under the suspicion of PC of unknown origin. Laparoscopy revealed multiple white nodules and masses on the surfaces of the peritoneum, mesentery, and intestinal serosa (Figure 2a, b). Ascites was also observed in the pelvic cavity (Figure 2b). Histopathological examination of the peritoneal biopsy specimen revealed diffuse proliferation of medium-sized cells with an increase in the nuclear: cytoplasmic ratio. The cells were relatively uniform in size and had hyperchromatic nuclei, without conspicuous nucleoli. Coarse nuclear chromatin and marked nuclear pleomorphism, which are often observed in typical DLBCL, were not prominent (Figure 3a, b). Immunohistochemically, these cells stained positive for CD10, 20, BCL6, and MYC and negative for CD3, CD5, CD23, CD30, BCL2, MUM1, and CyclinD1 (Figure 3c, d). The Ki-67 index of the tumor cells was approximately 95% (Figure 3e). EBV encode small RNA *in-situ* hybridization (ISH) was negative. Chromosomal analysis by G-banding was not performed. The histopathological diagnosis was HGBL, and required confirmation via cytogenetic analysis. Fluorescence ISH (FISH) of formalin-fixed paraffin-embedded tissues was used to detect *MYC*, *BCL2*, and *BCL6* gene rearrangements. The *MYC* gene status was analyzed using the Vysis LSI-MYC dual color break-apart rearrangement probe (Abbott Laboratories, Des Plaines, IL, USA) and Vysis LSI IGH/MYC dual fusion probe (Abbott). The *MYC* break-apart probe kit contains the LSI-MYC SpectrumOrange and LSI-MYC SpectrumGreen probes. The

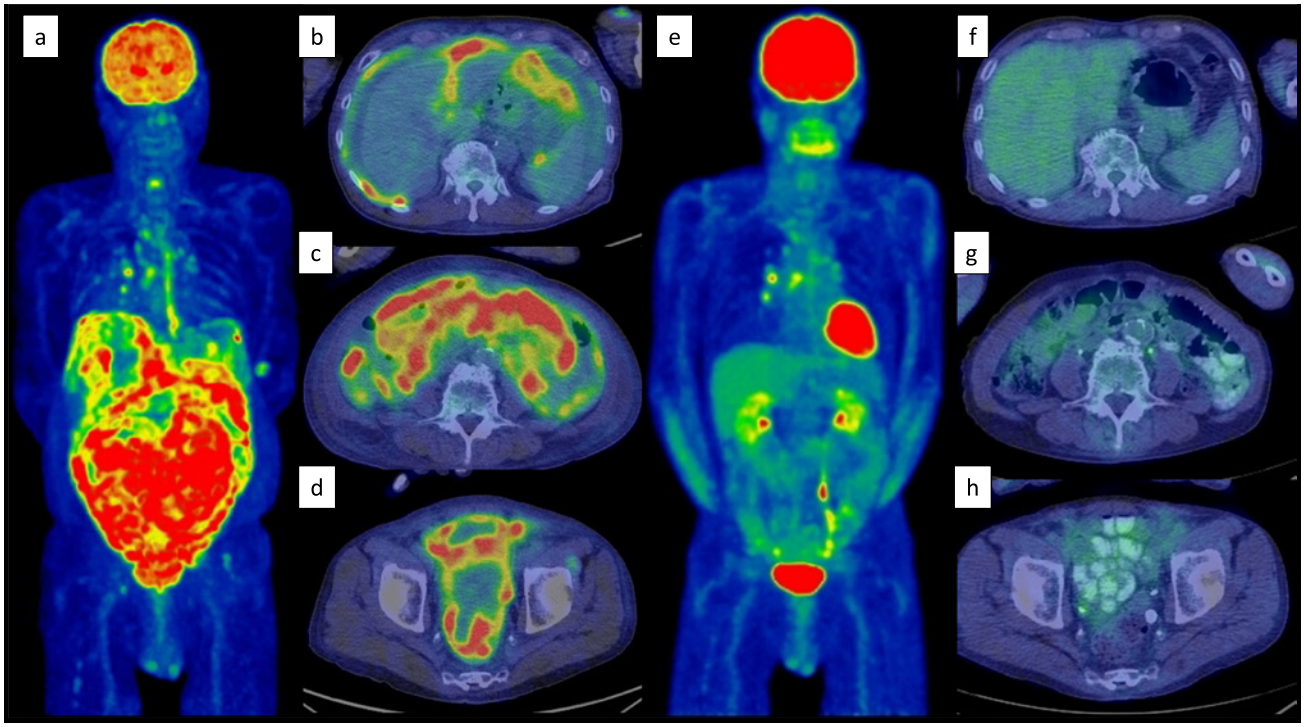


**Fig. 1.** Positron emission tomography-computed tomography (PET-CT) findings: (***a***–***d***) at the initial diagnosis and (***e***–***h***) after DA-EPOCH-R therapy completion. PET-CT at the initial diagnosis shows diffuse accumulation of $^{18}$F-fluorodeoxyglucose (FDG) in the thickened peritoneum, omentum, and mesentery with a maximum standardized uptake value of 9.8. No lymphadenopathy, hepatosplenomegaly, or solid organ lesions are observed (***a***–***d***). Mild FDG accumulation detected in the mediastinal lymph node (SUV max = 5.1), suggestive of reactive lymphadenopathy (***a***). Increased $^{18}$F-FDG uptake in the lower esophagus is also visible, compatible with esophagitis (***a***). All abnormal findings associated with PL are resolved (***e***–***h***).

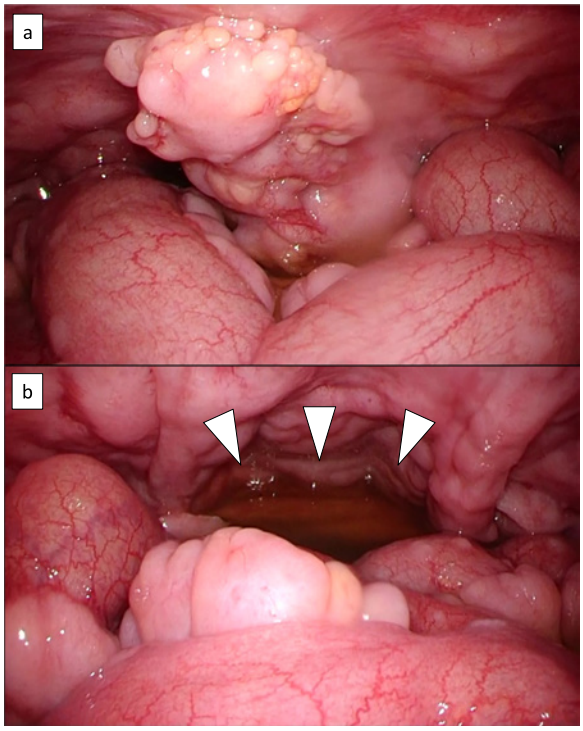


**Fig. 2.** Laparoscopy findings: Multiple white nodules and masses on the surfaces of the peritoneum, mesentery, and intestinal serosa are observed (***a***, ***b***). A small amount of ascites is also visible (white arrow head) (***b***).

LSI-MYC SpectrumOrange probe begins 119 kb centromeric to the 5' of the *MYC* gene and extends 260 kb towards the centromere. The LSI-MYC SpectrumGreen probe starts approximately 1.5 Mb telomeric to the 3' of *MYC* gene and extends towards the telomere for about 400 kb. The Vysis LSI IGH/MYC dual fusion probe kit contains an approximately 821 kb SpectrumOrange probe and an approximately 1.6 Mb SpectrumGreen probe, which cover the *MYC* and *IGH* regions, respectively. The *MYC* break-apart probe showed an extra red signal besides two sets of red and green fusion signals, in 84.0% of the cells (Figure 4a). The *IGH-MYC* dual-fusion probe showed no fusion signal; however, three isolated red signals and two green signals were observed, in most of the analyzed cells (Figure 4b). FISH analyses of both *BCL2* and *BCL6* rearrangements yielded negative results. Analyses of *MYC-IGK* and *MYC-IGL* translocations could not be performed at our institution. These results indicated a gain of the *MYC* gene accompanied by a green signal loss. Owing to the diagnosis of aggressive B-cell lymphoma, two cycles of rituximab plus miniCHOP (R-miniCHOP) therapy were administered, which resulted in lymphoma remission. Based on the additional cytogenetic analysis, we diagnosed the patient with HGBL, NOS, according to the revised fourth edition of the WHO classification, and the treatment regimen was switched to dose-adjusted (DA)-EPOCH-R therapy. Finally, the patient completed six courses of DA-EPOCH-R therapy, and complete metabolic remission was confirmed using PET-CT (Figure

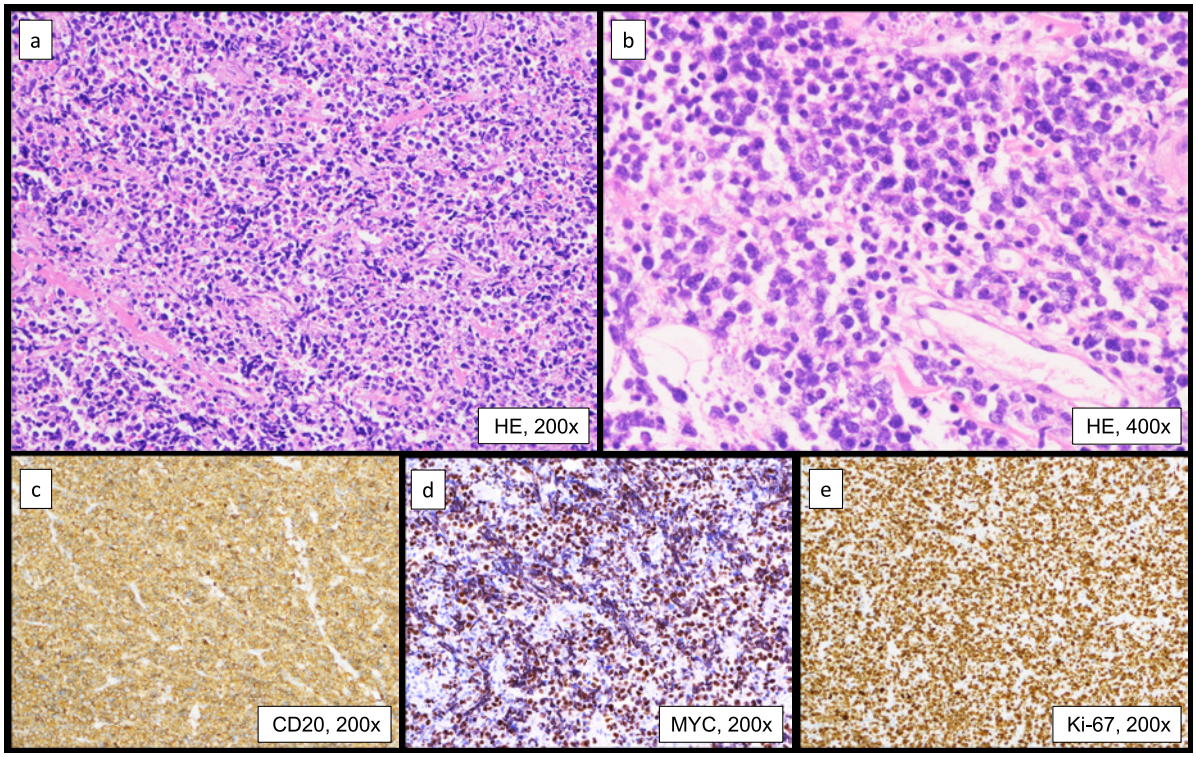


**Fig. 3.** Histopathological examinations of a peritoneal biopsy specimen: (***a***) hematoxylin and eosin staining (H&E) ×200, (***b***) H&E ×400, (***c***) CD20 ×200, (***d***) MYC ×200, (***e***) Ki-67 labeling index ×200. Diffuse proliferation of medium-sized cells with an increase in the nuclear: cytoplasmic ratio is observed. These cells are relatively uniform in size and have hyperchromatic nuclei without conspicuous nucleoli. Coarse nuclear chromatin and marked nuclear pleomorphism, which are often observed in typical DLBCL, are not prominent (***a***, ***b***). Tumor cells are positive for CD20 and MYC (***c***, ***d***). The Ki-67 labeling index is approximately 95% (***e***).

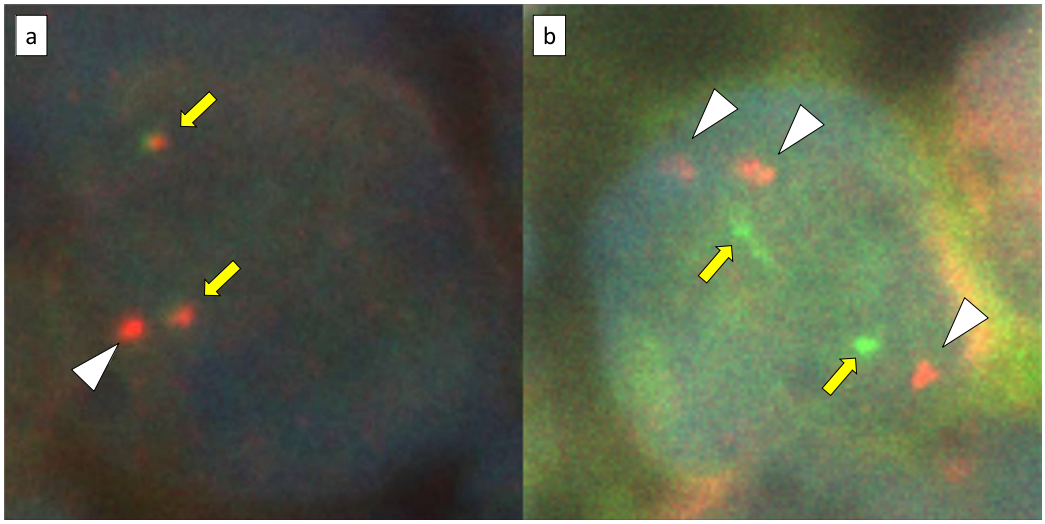


**Fig. 4.** *MYC* gene status analysis by fluorescence in situ hybridization using formalin-fixed paraffin-embedded tissues: (***a***) the Vysis LSI-MYC dual color break-apart rearrangement probe (Abbott Laboratories), which contains LSI-MYC SpectrumOrange and LSI-MYC SpectrumGreen probes. The LSI-MYC SpectrumOrange probe begins 119 kb centromeric to the 5' of the *MYC* gene and extends 260 kb towards the centromere. The LSI-MYC SpectrumGreen probe starts approximately 1.5 Mb telomeric to the 3' of *MYC* gene and extends towards the telomere for about 400 kb. (***b***) the Vysis LSI IGH/MYC dual fusion probe (Abbott Laboratories), which contains an approximately 821 kb of the SpectrumOrange probe and an approximately 1.6 Mb of the SpectrumGreen probe, which cover the *MYC* and *IGH* regions, respectively. The *MYC* break-apart probe shows an extra red signal (white arrow head) besides two sets of red and green fusion signals (yellow arrow) in 84.0% of the cells (***a***). The *IGH-MYC* dual-fusion probe shows no fusion signal, however, three isolated red signals (white arrow head) and two green signals (yellow arrow) are observed in most analyzed cells (***b***).

1e–h). Currently, the patient remains without recurrence for two years after completing chemotherapy.

## DISCUSSION

In the present case, because radiological findings, including PET-CT, provided only a suspected diagnosis of PC of unknown origin, exploratory laparoscopy was performed immediately. Consequently, a diagnosis of PL was made, and the patient received appropriate chemotherapy, which resulted in complete remission. Although no specific radiological findings to confirm the diagnosis of PL existed, characteristic CT findings of PL have been reported.[11,12] The frequencies of the peritoneal, omental, and mesenteric thickness in PL cases were 91–94%, 67–95%, and 67%, respectively.[11,12] In addition, lymphadenopathy, hepatosplenomegaly, digestive tract wall thickening, ascites, pleural effusion, and solid organ involvement were detected in 63–77%, 27–69%, 67%, 75–82%, 64%, and 56% of PL cases, respectively.[11,12] Regarding a differential diagnosis based upon the above radiological findings, the following disorders should be considered: PL, PC, peritoneal sarcomatosis, tuberculosis peritonitis, pseudomyxoma peritonei, and mesothelioma.[3,4,13] As PC is the most common, some helpful features for distinguishing PL from PC have been reported.[5,12] First, peritoneal lesions of PL are larger and more homogenous on contrast-enhanced CT than those of PC.[5] Second, PL often shows diffuse lymphadenopathy, whereas those of PC are usually located around the primary tumor.[5] Finally, splenomegaly is noted more frequently in PL patients (27%) than that in PC patients (2%).[12] In the present case, although diffuse thickening of the peritoneum, omentum, and mesentery was observed, neither lymphadenopathy or splenomegaly were not noted. That is, no typical CT findings suggestive of PL were noted, which required a diagnosis based on histopathological findings.

Since 2001, 45 PL cases have been reported in detail in the English-language literature. A summary of the 46 cases, including this case, is presented in Table 1.[14–52] The median age at disease onset was 55 years, with a male predominance. The major histopathological types included DLBCL in 20 cases, Burkitt's lymphoma (BL) in 11, follicular lymphoma in 3, and BL-like lymphoma in 2. Approximately 83% of cases (38/46) manifested an aggressive phenotype. Lymphadenopathy was observed in 16 cases (34.8%) and was primarily restricted to the intraperitoneal cavity (14 cases; 30.4%). Moreover, digestive tract infiltration was observed in 12 cases (26.1%): 6 in the stomach, 6 in the small intestine, and 1 in the colon. Splenomegaly was observed in only 7 cases (15.2%) (Table 1).

The rarity of PL may be contributed to by the absence of lymphoid tissues in the peritoneum.[4] Regarding the primary site of PL, a substantial number of cases may originate from the gastrointestinal epithelium. As described above, 26.1% of PL cases exhibit digestive wall infiltration (Table 1), and a previous report described that approximately two-thirds of PL cases exhibit bowel wall thickening.[11] The pathway by which lymphoma cells invade the peritoneum has not clearly elucidated. In cases of gastrointestinal lymphoma, possible

**Table 1.** A summary of reported cases of peritoneal lymphomatosis since 2001

| | n | % |
|---|---|---|
| Case number | 46 | 100 |
| Age, years | | |
| Median | 55 | |
| Min-max | 4–89 | |
| Gender | | |
| Man | 35 | 76.0 |
| Woman | 11 | 24.0 |
| Pathology | | |
| Diffuse large B-cell lymphoma | 20 | 43.5 |
| Burkitt lymphoma | 11 | 23.9 |
| Burkitt-like lymphoma | 2 | 4.3 |
| High-grade B-cell lymphoma, NOS | 1 | 2.2 |
| High-grade T-cell lymphoma | 1 | 2.2 |
| Plasmablastic lymphoma | 1 | 2.2 |
| B lymphoblastic lymphoma | 1 | 2.2 |
| T lymphoblastic lymphoma | 1 | 2.2 |
| Follicular lymphoma | 3 | 6.5 |
| B-cell lymphoma | 3 | 6.5 |
| T-cell lymphoma | 2 | 4.3 |
| Sites of involvement | | |
| Lymphadenopathy | 16 | 34.8 |
| Intraperitoneal lymphadenopathy | 14 | 30.4 |
| Splenomegaly | 7 | 15.2 |
| Digestive tract | 12 | 26.1 |
| Stomach | 6 | 13.0 |
| Small intestine | 6 | 13.0 |
| Large intestine | 1 | 2.2 |

pathways have been presumed, including the gastrocolic ligament, transverse mesocolon, and visceral peritoneal surface.[4,5] Notably, in the present case, lymphadenopathy, hepatosplenomegaly, gastrointestinal lesions, or a solid organ mass, which could be the origin of the PL, were not identified by detailed examinations using PET-CT and gastrointestinal fiberscopy. Thus, isolated peritoneal, omental, and mesenteric lesions were the only and primary lesions of PL. Among the 46 cases listed in Table 1, 13 presented with no lymphoma lesions other than PL.[16,24,27,29,33,37,38,43–45,49] All 13 cases, including the present case, are listed in Table 2. However, it should be considered that not all of the 13 cases underwent sufficient examinations, probably due to their poor general condition or regional differences (Table 2). In particular, PET-CT was performed in only four cases, and gastrointestinal fiberscopy results have not been described, except in the present case (Table 2). All patients exhibited an aggressive phenotype (Table 2). Eight patients received CHOP-based therapy and one patient received EPOCH therapy as induction therapy; however, lymphoma remission was observed in only six patients (Table 2). Six of the 13 patients died due to disease progression or tumor lysis syndrome (Table 2). The poor prognosis of primary PL may be attributed to its aggressive clinical course and difficulty in diagnosis owing to the absence of lymphadenopathy, hepatosplenomegaly, gastrointestinal lesions, and solid organ involvement. Reports regarding the origin of the lymphoma cells in patients with primary PL are lacking. It is speculated that in such cases, the primary lesion may spontaneously regress before PL development, or that physicians may fail to detect the primary lesion owing to insufficient examinations.

In the present case, FISH analysis demonstrated an increase in *MYC* copy number, whereas *BCL2* and *BCL6* rearrangements were not detected. As the histopathological findings indicated HGBL rather than DLBCL or BL, we diagnosed the patient with HGBL, NOS, accompanied by a gain of the *MYC* gene. The diagnosis of HGBL, NOS, is based on the diagnostic category of the revised fourth edition of the WHO classification, and this diagnosis is also applied in the fifth edition of the WHO classification published in 2022.[7,8] Regarding the FISH analysis of the present case, the break-apart rearrangement probe for *MYC* gene showed an unbalanced pattern of an extra red signal, in addition to, two fusion signals. This pattern denotes gain of the region covered by the red probe and deletion of the telomeric region covered by the green probe. Collinge *et al.* analyzed the 531 DLBCL and HGBL cases in which FISH *MYC* break-apart probe showed unbalanced patterns.[53] In that report, they detected the breakpoint locations and *MYC*-rearranged partners by using whole genome or capture sequencing.[53] In nine cases of loss of green signal, the breakpoint location was telomeric to *MYC* in all of the cases, namely, the *MYC* gene was located on the derivative chromosome containing the red signal.[53] In addition, 64% of them had various *MYC*-rearrangement partners, including *PAX5* or *BCL6* (29%), *IGL* or *IGK* (14%), *IGH* (7%), and other non-recurrent partners (14%).[53] Thus, the present case had an extra copy of the *MYC* gene in the region covered by the red probe, and the amplified *MYC* gene might have rearranged to some genes other than *IGH*.

Two cycles of R-miniCHOP therapy were initiated based on the initial histopathological diagnosis of aggressive B-cell lymphoma. Furthermore, based on the histopathological and cytogenic analysis results, the treatment regimen was changed to more intensive DA-EPOCH-R therapy. Currently, DA-EPOCH-R therapy is utilized in elderly patients with BL, with promising outcomes.[54] In addition, this regimen induced durable remission in 53 patients with DLBCL and HGBL with *MYC* rearrangement, in a prospective multicenter phase 2 study.[55] Recently, Zayac *et al.* reported a multi-institutional retrospective study of 160 patients with HGBL, NOS, in which *MYC* rearrangement, and *MYC* extra copies were observed in 28%, and 11% of the patients, respectively.[6] DA-EPOCH-R therapy was used in up to 43% of the patients as a remission induction therapy.[6] The prognosis of the patients with *MYC* rearrangement or *MYC* extra copies was not statistically inferior to that in patients without *MYC* alteration, irrespective of treatment regimens, including DA-EPOCH-R.[6] In the present case, as the histopathological diagnosis is HGBL, NOS, and the tumor had a possibility of *MYC* rearrangement, indication of DA-EPOCH-R therapy was appropriate. DA-EPOCH-R therapy was effective and well tolerated, resulting in long-term remission of the lymphoma.

**Table 2.** Reported cases of primary peritoneal lymphomatosis

| Case | Age | Sex | Pathological Diagnosis | PET-CT | Gastrointestinal Fiber Scope | Diagnostic Procedure | Treatment | Outcome | Reported year | Ref |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 71 | Male | High grade T-cell lymphoma | ND | ND | Autopsy | Not done | Dead by lymphoma | 2009 | 16 |
| 2 | 38 | Male | Burkitt-like lymphoma | Done | ND | Omentum, biopsy, US-guided | Unknown | Unknown | 2012 | 24 |
| 3 | 12 | Male | Burkitt lymphoma | Done | ND | Biopsy | Chemotherapy | Remission | 2013 | 27 |
| 4 | 57 | Female | DLBCL | ND | ND | Peritoneum, biopsy, laparoscopy | Not done | Dead by lymphoma | 2014 | 29 |
| 5 | 67 | Female | DLBCL | ND | ND | Peritoneum, biopsy, CT-guided | R-CHOP/MA | Remission | 2016 | 33 |
| 6 | 46 | Male | DLBCL | ND | ND | Peritoneum, biopsy, CT-guided | R-CHOP/MA | Remission | 2016 | 33 |
| 7 | 61 | Male | DLBCL | ND | ND | Omentum/Peritoneum, biopsy, US-guided | R-CHOP/ DA-EPOCH-R | Dead by lymphoma | 2019 | 37 |
| 8 | 62 | Male | DLBCL | ND | ND | Omentum, biopsy, US-guided | R-CHOP | Remission | 2019 | 38 |
| 9 | 16 | Male | DLBCL | ND | ND | Omentum, biopsy, laparoscopy | R-CHOP | Dead by lymphoma | 2021 | 43 |
| 10 | 74 | Male | DLBCL | ND | ND | Peritoneum, biopsy, US-guided | R-CHOP | Dead by TLS | 2022 | 44 |
| 11 | 63 | Male | DLBCL | Done | ND | Chest wall, biopsy | EPOCH-R | Remission | 2022 | 45 |
| 12 | 16 | Male | Burkitt lymphoma | ND | ND | Omentum/Peritoneum, biopsy, laparoscopy | CHOP | Dead by lymphoma | 2022 | 49 |
| 13 | 82 | Male | HGBL, NOS | Done | Done | Peritoneum, biopsy, laparoscopy | R-CHOP/ DA-EPOCH-R | Remission | Current case | |

• Primary peritoneal lymphomatosis indicates that the absence of lymphoma lesions other than peritoneal lymphomatosis.
• Abbreviations: DLBCL, diffuse large B-cell lymphoma; HGBL, high-grade B-cell lymphoma; NOS, not otherwise specified; PET-CT, Positron emission tomography-computed tomography; ND, not described; US, ultrasonography; CT, computed tomography; MA, methotrexate and cytarabine; TLS, tumor lysis syndrome; Ref, reference.

The prognosis of PC is generally poor, whereas remission can be induced in PL with appropriate chemotherapy because most PL manifest as aggressive phenotypes. Several PL case reports described initial misdiagnosis of PC rather than PL, which could be a risk factor for delay in initiating chemotherapy, leading to treatment failure.[14,20,26,32,43] In particular, as described above, the diagnosis of primary PL might be more difficult and all case reports had aggressive phenotypes. PL should be considered in the differential diagnosis of peritoneal lesions, even in the absence of other lymphoma lesions. Furthermore, subsequent prompt histopathological examinations and appropriate chemotherapy, such as DA-EPOCH-R therapy, lead to better patient outcomes.

## CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

## ACKNOWLEDGMENTS

None.

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