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Case report

Long-term remission in a patient with NK/T cell intravascular lymphoma with autologous hematopoietic cell transplantation

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Intravascular lymphoma (IVL) is a rare subtype of lymphoma, mostly of B-cell origin. A few cases of IVL have been reported as having NK/T cell origins (IVNKTL). These cases are known to be fatal, especially when systemic symptoms are present. We report the case of a patient of IVNKTL who was refractory to initial treatment and received autologous hematopoietic stem cell transplantation (auto-HSCT). She has maintained complete remission (CR) for over eight years. Our case might support the evidence of auto-HSCT for the treatment of IVNKTL with chemosensitivity.

Keywords: intravascular lymphoma, intravascular NK/T cell lymphoma, hematopoietic stem cell transplantation

INTRODUCTION

Intravascular lymphoma (IVL) is a rare form of lymphoma.[1] Most tumor cells are of B-cell origins (IVBCL),[2–4] however, there are also reports of IVL of NK/T cell origins (IVNKTL). In the last World Health Organization (WHO) classification of lymphoid neoplasms, IVNKTL is recognized as a close entity to extranodal NK/T-cell lymphoma (ENKTL).[5] The treatment strategy of IVNKTL remains unknown, especially when systemic symptoms such as fever, hepatosplenomegaly, and pancytopenia is present.

In this report, we experienced a case of IVNKTL under remission with autologous hematopoietic stem cell transplantation (auto-HSCT).

CASE PRESENTATION

A 58-year-old female without any previous medical history presented to our hospital complaining general fatigue for over six months. On admission, all physical examinations were normal, except for the presence of periorbital edema. Her complete blood count (CBC) was White blood cells 6,000/μL (Myelocytes 0.5%, Neutrophils 76%, Lymphocytes 6.5%, Monocytes 17%), Hemoglobin 9.2 g/dL, and Platelets 160,000/μL. Her serum laboratory results were Lactate dehydrogenase (LDH) at 1135 U/L, Aspartate aminotransferase (AST) at 69 U/L, Alanin aminotransferase (ALT) of 42 U/L, C-reactive protein (CRP) at 12.91 mg/dL, Ferritin at 1279.1 ng/mL, and Soluble IL-2 receptor (sIL-2R) levels of 4698 U/mL. All blood test results are shown in Table 1. Hepatosplenomegaly was detected on a whole body Computed Tomography (CT) scan without lymphadenopathy (Figure 1). Her fever rose to 40°C and she became less responsive to stimuli early after admission. Her spinal fluid exam was within the normal range, and brain Magnetic Resonance Imaging (MRI) scans revealed no abnormalities. Hemophagocytes were identified on a bone marrow exam. At that point she fulfilled the hemophagocytic lymphohistiocytosis (HPS/HLH) diagnostic criteria.[6] IVL was considered as one of the causes of HPS/HLH, we performed a random skin biopsy. As her condition rapidly worsened, we began to treat her with dexamethasone, cyclosporine, and etoposide, according to the HLH 2004 protocol[7] on the fourth day of her hospital stay. She began to respond to the treatment, becoming afebrile, and her blood count gradually improved. After an infusion of a third course of etoposide on the 10th day of her treatment, her biopsy exam revealed that large cells with prominent nucleoli were lodged in the lumina of small sized vessels, indicating IVL (Figure 2). Because she was complicated by HPS, we began an etoposide (50 mg/m^2 on days 1–4), prednisone (50 mg/m^2 on days 1–5), vincristine (0.4 mg/m^2/d on days 1–4), cyclophosphamide (750 mg/m^2/d on

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Table 1. Laboratory findings on admission

Complete blood count		
White blood cell	6,000	/μL
Myelocyte	0.5	%
Neutrophil	76	%
Eosinophil	0	%
Basophil	0	%
Monocytes	17	%
Lymphocyte	6.5	%
Red blood cell	3.05×10^{8}	/μL
Hemoglobin	9.2	g/dL
Hematoclit	27.5	%
MCV	89.9	fL
Platelet	1.6×10^{6}	/μL

Coagulation test		
Prothrombin time	10.9	s
APTT	28.7	s
FDP	10.5	ug/mL
Fibrinogen	263	mg/dL

Biochemstry		
Albumin	2.5	g/dL
Lactate dydrogenase	1,135	U/L
Aspartate aminotransferase	69	U/L
Alanin aminotransferase	42	U/L
Total bilirubin	0.7	mg/dL
Uric acid	2.7	mg/dL
Blood Urea Nitrogen	17	mg/dL
Creatinin	0.69	mg/dL
C-reactive protein	12.91	mg/dL
Natrium	124	mEq/L
Potassium	4.9	mEq/L
Chloride	94	mEq/L
Calcium	8.2	mg/dL
Iron (Fe)	18	ug/dL
Total iron biding capacity	264	ug/dL
Ferritin	1,279.1	ng/mL
Glucose	115	mg/dL
Triglyceride	278	mg/dL

Immunologic test		
Immunoglobulin G (IgG)	1,156.2	mg/dL
Immunoglobulin A (IgA)	286.1	mg/dL
Immunoglobulin M (IgM)	30.5	mg/dL
Soluble IL-2 receptor	4,698	U/mL

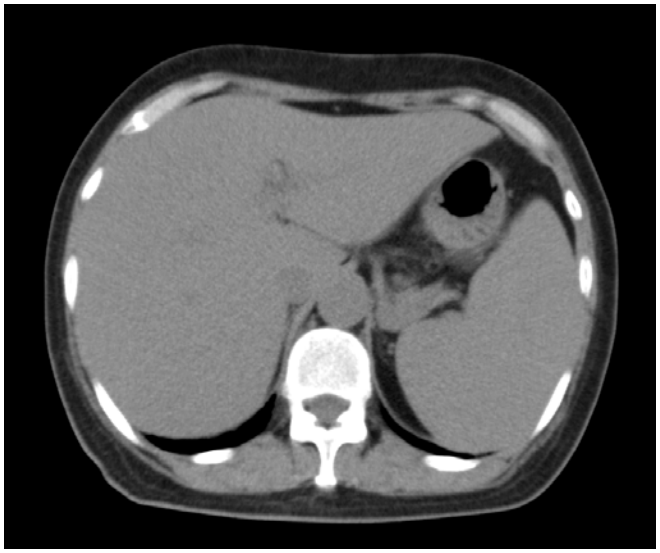

Fig. 1.
CT scan image shows an enlarged spleen.

day 5), and doxorubicin (10 mg/m^2/d on days 1–4) (EPCOH) regimen. Her immunostaining profile revealed that the large tumor cells were positive for CD3, CD56, and EBV-encoded small nuclear RNA (EBER), and negative for CD20 and CD79. Ki67 positivity was > 90% (Figure 3). This finding indicated that her diagnosis was IVNKTL. As a result, she was started on a dexamethasone (33mg /body, on days 2-4), methotrexate (2,000 mg/m^2 on day 1), ifosfamide (1.5 g/m^2 on days 2–4), l-asparaginase (6,000 U/m^2 on days 3–9), and etoposide (100 mg/m^2 on days 2–4) (SMILE) regimen. She achieved complete remission (CR) after four cycles of SMILE treatment with resolution of clinical symptoms and normalization of clinical laboratory tests. However, after two months her LDH and sIL-2R levels became elevated and she was diagnosed as having a relapsed disease. We responded by conducting four courses of gemcitabine (1000 mg/m^2 on days 1 and 8), dexamethasone (20 mg/d on days 1–4 and 11–14), and cisplatin (25 mg/m^2 on days 1–3) (GDP) as salvage chemotherapy. She then received auto-HSCT (conditioning: ranimustin (200 mg/m^2 on days before auto-HSCT 8 and 3), carboplatin (300 mg/m^2 from days before auto-HSCT 7 to 4), etoposide (500 mg/m^2 from days before auto-HSCT 6 to 4) and cyclophosphamide (50 mg/kg on days before auto-HSCT 3 and 2) (MCEC)) and remained in CR for over eight years.

DISCUSSION

We reported a case of IVNKTL under long-term remission with auto-HSCT.

IVL is a rare subtype of lymphoma with intravascular invasion, which is known to show a rapid clinical course.

IVL is often difficult to diagnose because it does not form a masse, unlike ordinary lymphoma.[3] Positron Emission Tomography (PET)-CT is often used to determine the site of biopsy, as it often shows a wide accumulation in the bone marrow, liver, and lungs.[8] In our case, a patient was admitted to the hospital as an emergency and there was no time to perform PET-CT. However, the patient had hepatic dysfunction at the time of initial presentation, so we expected to see accumulation in the liver.

IVL is most often of B-cell origin,[1] and IVNKTL is considered to be the rarest form of lymphoma and to date, 40 cases have been reported (Table 2).

IVNKTL is confirmed by two criteria: first, small vessels must be filled with atypical cells with large nuclei; and second, NK/T cell markers such as CD56 and CD3 must be present. Atypical cells are also often EBER positive.[12,17] In 5th edition of WHO classification, IVNKTL is considered a form of

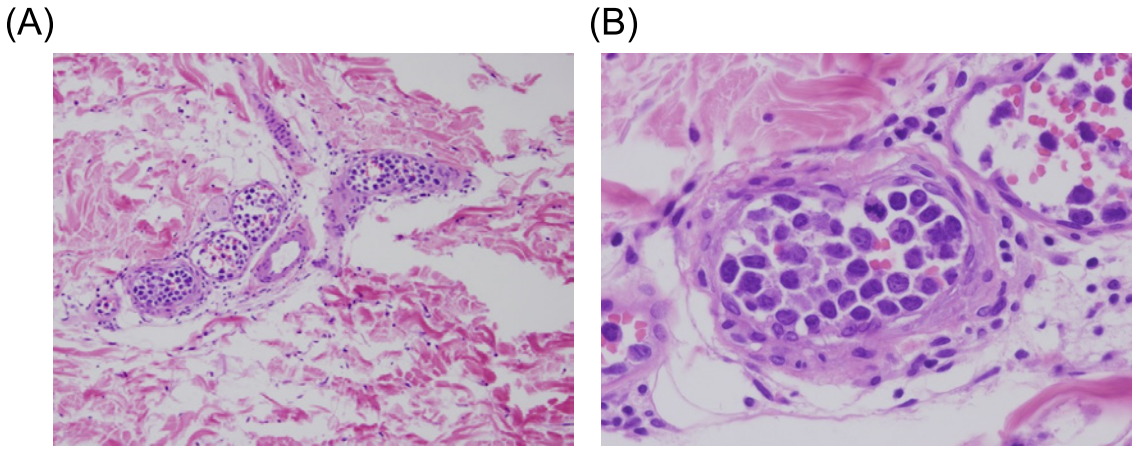

Fig. 2.
Dilated vessels with microthrombi and infiltrated by medium-sized irregular blast-like cells with hyperchromatic nuclei and prominent nucleoli. HE (×40) (***A***), HE (×200) (***B***).

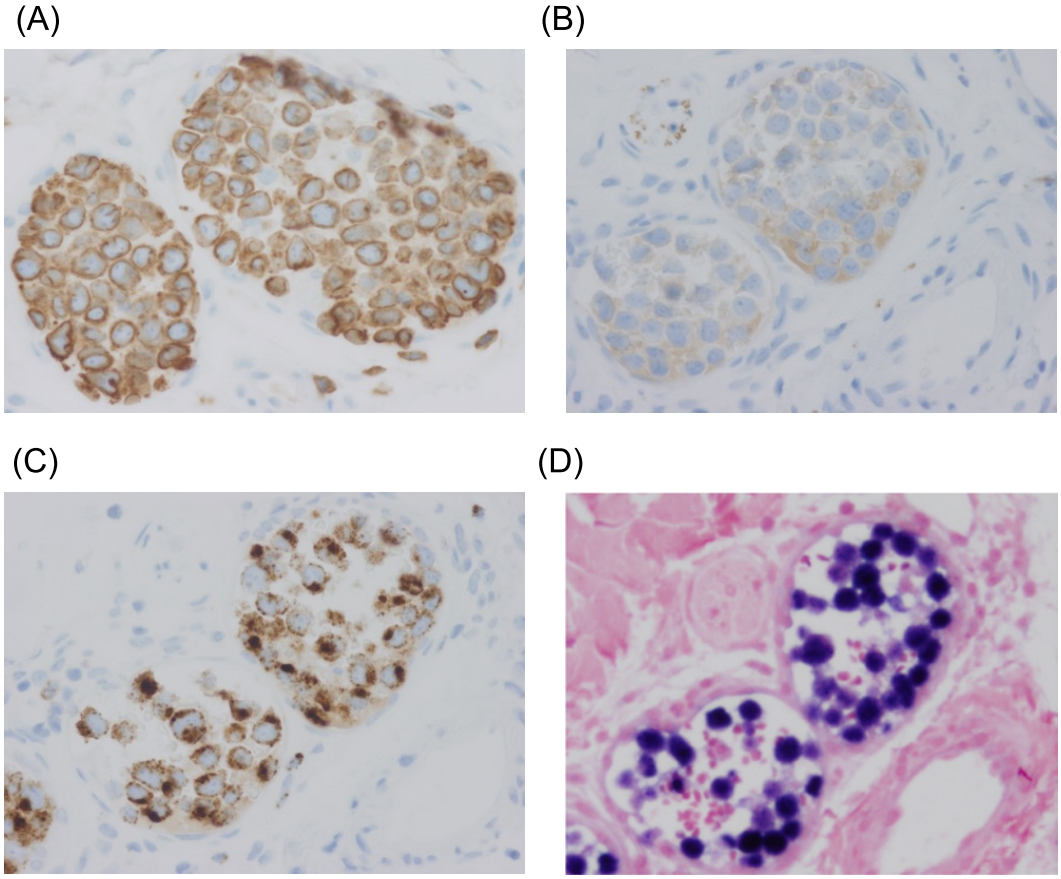

Fig. 3.
Immunohistochemistry with CD3 (***A***), CD56 (***B***), granzyme B (***C***) and EBER (***D***) demonstrate a positive staining of the neoplastic intravascular cells.

ENKTL and shows a preference for skin and central nervous system.[5] According to previous reports on IVNKTL, its prognosis is dismal. Chemotherapies such as Cyclophosphamide, Doxorubicin, Vincristine, and Prednisolone (CHOP) are often used. Some patients with IVNKTL show longer survivals of more than one year when the tumors are limited to the skin. However, those with fever and liver dysfunction, likely complicated by systemic infiltration of tumor cells, have been reported to have fatal courses (Table 2).

L-asparaginse containing regimens, such as SMILE, have significantly improved the outcome of NK/T cell lymphoma and are now the most commonly employed as an initial ther-

Table 2.

Case	age/sex	Clinical features	IHC	EBV	Treatment	Outcome	Reference
1	72/M	Erythema, Confusion	CD3, CD56, GrB, TIA-1	+	chemotherapy	Died, 7M	9
2	45/M	Erythema, Fever	CD3, CD30, CD56, GrB, TIA-1	+	No treatment	Died, 2W	10
3	52/F	Erythema, Fever	CD3, CD30, CD56, GrB, TIA-1	+	CHOP	Died, 6M	10
4	32/M	Erythema, Fever	CD3, CD30, CD56, GrB, TIA-1	+	CHOP	Died, 4M	10
5	18/F	Erythema	CD3, CD30, CD56, GrB, TIA-1	+	CHOP	Alive,36M	10
6	51/M	Erythema, Fever	CD3, CD30, CD56, GrB, TIA-1	+	CHOP+VP16	Died, 6M	10
7	54/M	Fever, Liver dysfunction	CD3, GrB, CD56, Ki-67 80%	+	No treatment	Died, 3D	11
8	51/M	Erythema	CD3, CD8, TIA-1, GrB	+	unknown	unknown	12
9	79/M	Renal failure	CD3, GrB, CD56	+	No treatment	Died, 3W	13
10	41/M	Erythema	CD3, CD56, GrB, TIA-1, CD30, Ki-67 100%	+	CHOP, ASCT	Alive, 1Y	14
11	47/F	Fever, Arthralgia, Confusion	CD3, CD56, GrB, TIA-1, CD7, CD2	-	No treatment	Died, 1M	14
12	40/F	Erythma, Hemiplegaia	CD3, CD56,GrB, TIA-1 CD45, Ki-67 100%	+	CODOX-M/IVAC	Alive, 7M	15
13	63/M	Erythema, Fever, Confusion, Pancytopenia	CD3, CD56, TIA-1, CD45RO, CD2	+	No treatment	Died, 6M	16
14	63/M	Erythema	CD3, TIA-1, CD45RO, CD2	-	CHOP	Died, 7M	16
15	67/F	Erythema	CD3, TIA-1, CD45RO, CD2		No treatment	Died, 1W	16
16	87/M	Erythema	CD3, TIA-1, CD45RO, CD2	+	No treatment	Died, 1M	16
17	54/M	Erythema	CD3, CD56, GrB, TIA-1, CD30, Ki-67 100%	+	CHOP	Died, 17M	17
18	62/M	Erythema	CD3, CD30, CD56, GrB, TIA-1	+	CHOP, DHAP	Alive, 8M	18
19	38/F	Erythema, Fever	CD3, GrB, CD56, Ki-67 90%	+	CHOP	Died, 13M	19
20	47/F	Erythema	CD3, GrB	+	chemotherapy	Alive,18M	20
21	20M	Fever, Hepatosplenomegaly	CD3, GrB, CD56, Ki-67 90%	+	SMILE, ASCT	Alive, 18M	21
22	65/M	Erythema, Fever, Hepatosplenomegaly	CD3, GrB, CD56, Ki-67 90%	+	No treatment	Died, 1D	22
23	56/M	Nodules, Fever	CD3, GrB	+	CHOP	Died, 1Y	22
24	71/F	Erythema	CD3, CD56, GrB, TIA-1 Ki-67 99%	+	No treatment	Alive, 5M	23
25	23/F	Erythma, Leg edema, Fever	CD3, CD56, TIA-1	+	CHOP.etc, allo-HSCT	Died, 9M	24
26	42/F	Erythema	CD3, CD56, GrB, Ki-67 100%	+	CHOP, EPOCH, etc.	Alive, 14M	25
27	22/M	Erythema, Fever	CD3, CD56, GrB, Ki-67 100%	+	CHOP	Died, 2M	26
28	42/F	Erythema	CD3, CD56, GrB, Ki-67 100%	+	CHOP	Alive, 14M	26
29	18/F	Erythema	CD3, GrB, CD56, CD2, CD30, Ki-67 100%	+	Radiation	Alive, 1M	27
30	29/M	Erythema, Liver dysfunction	CD3, CD30, CD56, GrB	+	chemotherapy	Died, 3M	28
31	84/F	Erythema	CD3, CD30, CD56, GrB, TIA-1	+	No treatment	Alive, 4M	29
32	46/M	Confusion, Hedache	CD3, GrB, Ki-67 100%	+	No treatment	Died, 2M	30
33	57/M	Enlargement of testis	CD3, GrB, Ki-67 90%	+	CHOP+tumor resection	Alive 22M	31
34	38/M	Cough, Fever	CD3, GrB, CD56, TIA-1, Ki-67 100%	+	CHOP	Died, 2M	32
35	21/M	Cough, Fever	CD3, GrB, CD56, TIA-1, Ki-67 99%	+	No treatment	Died, 2M	32
36	23/M	Erythema, Fever	CD3, GrB, CD56, CD2, TIA-1, Ki-67 90%	+	P-GEMOX	Died, 18M	32
37	54/F	Erythema	CD3, GrB, CD56, CD2, TIA-1, Ki-67 95%	+	No treatment	Died, 3M	32
38	66/M	Lymphodenopathy	CD3, CD43, CD8, GrB, TIA-1, perforin	-	CHOP	Died, 2M	33
39	60/M	Erythema	CD3, GrB, CD56, Ki-67 80%	+	P-GEMOX	Alive, 18M	34
40	53/M	Confusion	CD3, GrB, CD56, Ki-67 100%	+	SMILE, HAM, allo-HSCT	Alive, 8Y	35

allo-HSCT allogenic hematopoietic stem cell transplantation; CHOP cyclophosphamide, doxorubicin, vincristine and prednisolone; CODOX-M cyclophosphamide, doxorubicin, vincristine, cytarabine and methotrexate; DHAP dexamethasone, cytarabine and cisplatin; EPOCH etoposide, cyclophosphamide, doxorubicin, vincristine and prednisolone; HAM cytarabine and mitoxantrone; IVAC ifosfamide, etoposide and cytarabine; P-GEMOX pegaspargase, gemcitabine and oxaliplatin; SMILE dexamethasone, methotrexate, ifosfamide, l-asparaginase, and etoposide.

apy. However, in approximately 20 to 40% of cases, SMILE is ineffective, and in these cases, there is no appropriate salvage and the prognosis is poor.[36–38] This might be applied to IVNKTL.

Recently, patient with IVNKTL refractory to SMILE regimen who achieved long-term (eight years) survival with allogeneic hematopoietic stem cell transplantation (allo-HSCT) was reported.[35] A randomized trial failed to prove the superiority of allo-HSCT over auto-HSCT in patients with peripheral T-cell lymphoma.[39] Additionally, the comparative outcome results for allo-HSCT and auto-HSCT in patients with NK T cell lymphoma who achieved CR prior to transplantation remain to be determined due to higher therapy related toxicity after allo-HSCT.[40,41]

The validity of auto-HSCT remains to be confirmed, our case indicates that auto-HSCT might achieve long-term survival for IVNKTL refractory to l-asparaginase containing regimen.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest regarding the publication of this paper.

AVAILABILITY OF DATA AND MATERIALS

The data used to support the findings of this study are included within the article.

CONTENT

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

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