



# Discordant lymphomas of classic Hodgkin lymphoma and peripheral T-cell lymphoma following dupilumab treatment for atopic dermatitis

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## Abstract

There have recently been a few case reports of cutaneous T-cell lymphomas following treatment of atopic dermatitis with dupilumab, which works binding to the interleukin (IL)-4 receptor and inhibiting the JAK/ STAT cascade located downstream of both IL-4 and IL-13. Here, we report the first case of Hodgkin lymphoma (HL) in a patient treated with dupilumab for one year. Based on multiple biopsies, this case was diagnosed as a rare combination of discordant lymphomas of HL and peripheral T-cell lymphoma. As both lymphomas are known to overexpress IL-13, future studies should carefully evaluate the effect of anti-IL-13 therapy. A literature review showed that dermatitis persisted or worsened in all reported lymphoma cases following dupilumab and cutaneous T-cell lymphoma was diagnosed within 2 years of the start of treatment with dupilumab. In these cases, with the addition of our own, the median interval was 12 months, and 31% needed multiple biopsies for diagnosis of lymphomas. Our results demonstrate a need to be alert to potential development of lymphomas associated with the IL-13 and IL-4 pathways in patients with poorly responsive atopic dermatitis receiving dupilumab, and to consider the possibility of composite or discordant lymphomas in diagnosis and treatment of lymphomas.

**Keywords** Dupilumab · Discordant lymphomas · Hodgkin lymphoma · Peripheral T-cell lymphoma · Atopic dermatitis

## Introduction

Dupilumab administered against atopic dermatitis or bronchial asthma works by binding to the alpha subunit of the interleukin (IL)-4 receptor and inhibiting the JAK/ STAT cascade located downstream of both IL-4 and IL-13 [1]. In vitro, blocking IL-13 or IL-4 pathway caused cyto-reduction of cases of cutaneous T-cell lymphoma (CTCL) cell lines which harbored overexpression [2]. Though a case with partial remission of CTCL by dupilumab therapy was

reported recently [3], it is noteworthy that a few reports have shown the development of T-cell lymphoma following treatment by dupilumab for atopic dermatitis [4–8], while the association between dupilumab and Hodgkin lymphoma (HL) has not been reported. Here, we report a case developing discordant lymphomas of HL and peripheral T-cell lymphoma, not otherwise specified (PTCL-NOS) after receiving dupilumab for a year.

## Case presentation

A 47-year-old male was introduced to our hospital for bulky mass from the right axilla to the chest wall, and scaly erythematous patches on the upper and lower limbs extensively with marked pigmentation (Fig. 1A). He had been suffered from atopic dermatitis and asthma since childhood and had undergone dupilumab administration for the last year. Positron emission tomography (PET) scan showed increased uptake of 18 F-fluorodeoxyglucose (FDG) in multiple lymph nodes (maximum standardized uptake value (SUV max) 22.8 at the right axilla), the spleen and multiple bones, and skin

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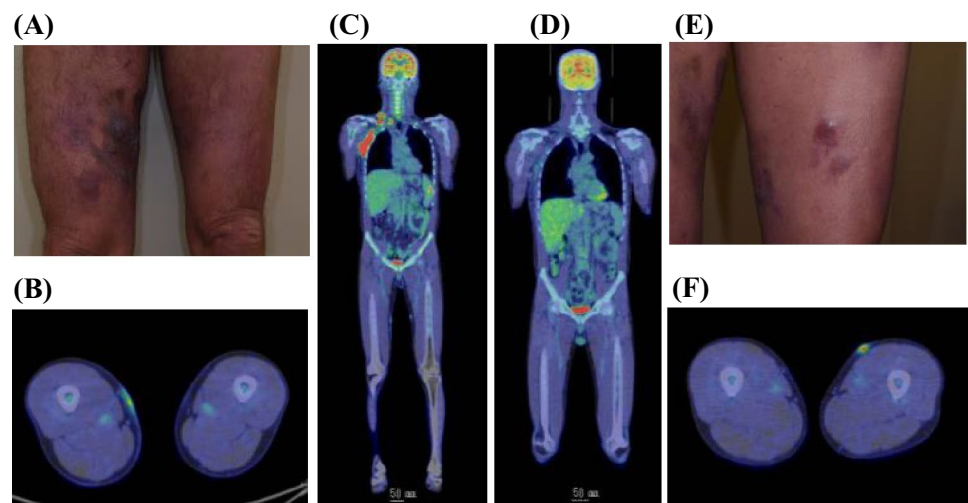
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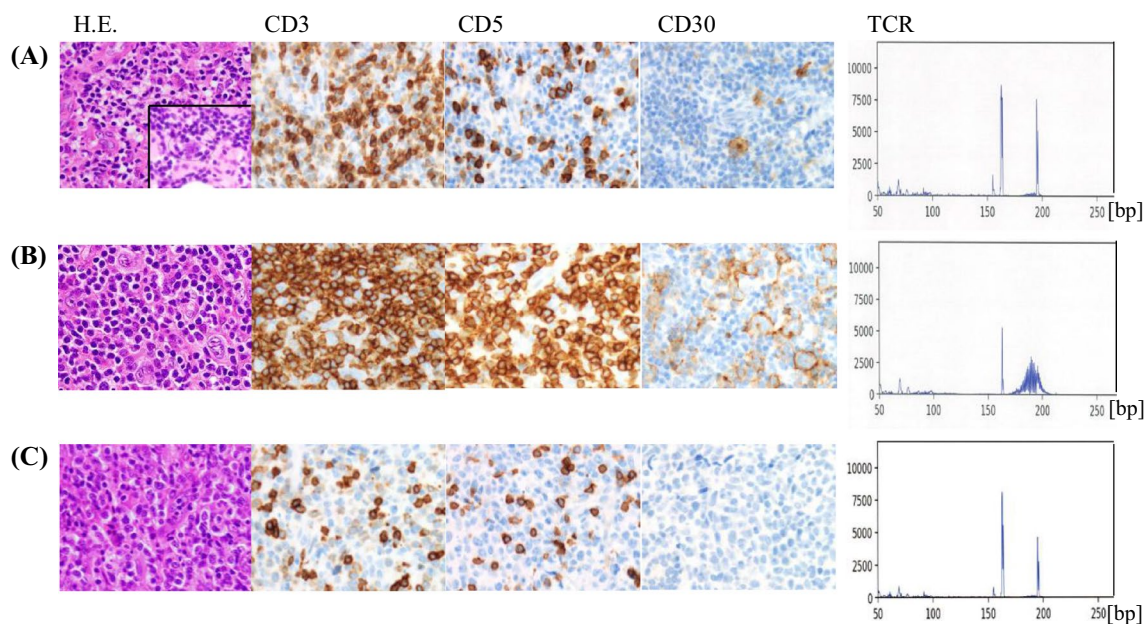
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**Fig. 1** The macroscopic findings of the skin lesions and the results of positron emission tomography (PET) scan. **A** scaly erythematous patches and pigmentations of the bilateral thighs at diagnosis, **B** and **C** PET scan images at diagnosis, **D** PET after two cycles of AAVD regimen, **E** the solid tumor in the skin of the left thigh at relapse, and **F** PET at relapse. **B** and **F** are PET images of the bilateral thighs



lesions (SUV max 4.4) (Fig. 1B, C). A needle biopsy of the skin of the right thigh revealed infiltration of lymphoma cells of T-cell origin, with a rearranged T-cell receptor (TCR) gamma chain (Fig. 2A). Immunohistochemistry revealed that cell surface marker of atypical T-cells was positive for CD3, CD4, and Granzyme B, partially positive for CD8, and negative for CD5, CD7, and CD20. On the other hand, a biopsy of the right axillary lymph node revealed classic HL of nodular sclerosis type (Fig. 2B). The structure of the lymph nodes has disappeared, and large Hodgkin cells and

Reed-Sternberg cells are proliferating in a scattered or sheet-like manner against the background of a large number of small lymphocytes, plasma cells and histiocytes accompanied by in or around the tumor. Although immunostaining of Pax5 was negative, Hodgkin and Reed-Sternberg (H/RS) cells positive for CD15, CD30, MUM-1 and PD-L1 were detected in the specimen of the lymph node. These H/RS cells were negative for CD3, CD5, CD7 and CD45, which indicated the diagnosis of the axillary lymphadenopathy was not T-cell lymphoma. The cytotoxic molecules TIA1 and



**Fig. 2** Pathology and rearrangement of T-cell receptors (TCR) at diagnosis and at relapse. The left four columns show microscopic images (H.E. stain, CD3, CD5, and CD30 immunostaining,  $\times 400$ , respectively) of the specimens of **(A)** the skin of the right thigh at diagnosis, **B** the right axillary lymph node at diagnosis, and **C** the

tumor of the left thigh at relapse. The panels of the right column show rearrangement of TCR. The panel in the lower right part of the leftmost panel **(A)** encloses Hodgkin-like tumor cells. *H.E.* Hematoxylin–eosin

Granzyme B were negative in the H/RS cells. Discordant or composite lymphomas comprised T-cell lymphoma and classic HL which are diagnosed simultaneously are rare and a review of the literature founded only one discordant and ten composite cases (Table 1) [9–15]. A few CD15- or CD30-positive cells and the cells that appeared to be Hodgkin cells were detected in the specimen of the skin (Fig. 2A). Therefore, we adopted a simpler hypothesis that HL cells infiltrated the skin with predominant reactive T-cells with cellular atypia, instead of discordant lymphomas, at this time.

He underwent six cycles of brentuximab-vedotin, doxorubicin, vinblastine, and dacarbazine (A-AVD) regimen [16], and both lymphadenopathy and the abnormal skin lesions improved initially, and almost all the uptake by FDG-PET scan disappeared after two cycles (Fig. 1D). Six months later, right after the sixth cycle, solid tumors developed in the skin of the left lower limb and the left upper arm (Fig. 1E, F). Re-biopsy of the mass in the left thigh revealed infiltration of T lymphocytes with even more severe atypia, leading to the diagnosis of PTCL-NOS (Fig. 2C). CD15 and CD30 were negative. Since it is sometimes difficult to distinguish HL and PTCL, clonality of the TCR gamma chain was evaluated using the three specimens: the right axillary lymph node and the skin taken before chemotherapy, and the skin tumor at relapse. Two skin specimens revealed the same clonal rearrangement of TCR indicated by two peaks by multiplex polymerase chain reaction (PCR) (Fig. 2A, C); on the other hand, the left peak was in common, while the same right peak representing the same TCR clonality as the skin lesions was not detected in the lymph node (Fig. 2B). Overall, we concluded that he had discordant lymphomas of HL and PTCL-NOS at the first diagnosis, and the component of PTCL persisted after A-AVD therapy.

## Discussion

To our knowledge, our case was the first Hodgkin lymphoma case following dupilumab therapy. A rare combination of discordant lymphomas, HL and PTCL, were detected in our case after dupilumab therapy, both of which are known to overexpress IL-13. Despite postulated anti-lymphoma activity of IL-13-blockade, some CTCL cases including mycosis fungoides (MF) have been reported developing after dupilumab (Table 2) [4–8]. IL-13 was overexpressed in CTCL with autocrine growth-promoting activity and simultaneous antibody-based neutralization of IL-13 and IL-4 inhibited *in vitro* proliferation of CTCL cells [2]. In a few reported CTCL cases, an initial response was obtained by administering dupilumab, however, worsening of the disease followed [3]. Blocking IL-13 or IL-4 pathway caused cytoreduction of CTCL cell lines, and initial response of the CTCL patients accorded with these experimental results.

However, in other cases, dupilumab showed minimal effect or even accelerated CTCL progression, casting doubt on the clinical usefulness of the anti-IL-13 therapy against CTCL [3, 7]. Though the underlying mechanism of the discrepancy between *in vitro* and *in vivo* activity of IL-13-blockade has been still obscure, increase in infiltrated atypical lymphoid cells was detected in the MF cases diagnosed after dupilumab therapy, which might be related to the change of balance of Th1/Th2 cytokine microenvironment and lead to the development of MF [6]. Chronic inflammation and infection are known to be related to some types of lymphomas such as extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue and Epstein-virus positive diffuse large B-cell lymphoma, and it was possible that chronic inflammation of atopic dermatitis contributed to exacerbation of lymphomas [17, 18]. Though the association of dupilumab use and the development of lymphomas in our case is difficult to prove, the *in vivo* effect of anti-IL-13 therapy should be cautiously determined in future studies.

Sokumbi et al. revealed that the time since initiating dupilumab for dermatitis to diagnosis of lymphoma was 9.8 months on average [6]. All the previously reported fifteen cases were diagnosed CTCL within 2 years (9.1 months on average) since start of dupilumab and had poor responses to the therapy or exacerbation of dermatitis during treatment (Table 2) [4–8]. Median interval was 12 months including this case, which was consistent with the clinical course of our case. We conducted three times of biopsy resulting in discordant lymphomas. Including the reported case and this case, 31% of sixteen cases required multiple biopsies before diagnosis of lymphomas after administration of dupilumab (Table 2). The role of inhibition of IL-13 pathway in the development of lymphomas has not been elucidated precisely yet, these suggest that there is a possibility of developing lymphomas in the cases with poor response to dupilumab and performing multiple biopsy evaluation can be needed for diagnosis of lymphomas. TCR was evaluated by multiplex PCR assay characterized by better sensitivity but poorer specificity than southern blotting assay and finding the common peaks in both skin specimens at diagnosis and at relapse led to the diagnosis of recurrence of T-cell lymphoma. The possibility of composite or discordant lymphomas should be considered while referring to the difference in accumulation in FDG scan.

Interestingly, the specimen of the second skin biopsy at relapse lacked expression of CD15 and CD30 although that of first skin biopsy at diagnosis revealed positivity of these markers. Brentuximab-vedotin, which is antibody–drug conjugate against CD30 positive cells, are effective for both CD30 positive Hodgkin lymphoma and PTCL, consistent with initial improvement of both lymphomas in our case [16, 19]. CD30 negative T-cell clones might have escaped from the immunochemotherapy and

**Table 1** The current and previously reported cases of discordant or composite lymphomas comprised of classic Hodgkin lymphoma and T-cell lymphoma

Case	Age, sex	Date (year)	Composite/discordant	Biopsy	Diagnosis	Immunostaining				PCR			References
						T-cell				Hodgkin RS cell			
						CD3	CD4	CD5	CD8	CD15	CD30	TCR	
1	60, M	2002	Discordant	Cervical LN	Classical HL (NS)	+	+	+	Weak	+	+	NA	[9]
2	76, F	2012	Composite	Inguinal LN/BM	PTCL	+	-	NA	+	-	+	+	[10]
3	19, M	2011	Composite	LN	Classic HL (MC), PTCL-NOS	+	-	NA	+	-	+	+	[10]
4	74, M	2012	Composite	LN	Classic HL (MC), PTCL-NOS	+	+	NA	-	+	+	+	[10]
5	43, M	2013	Composite	LN	Classic HL (MC), T-PLL	+	+	NA	+	+	+	+	[10]
6	57, F	2010	Composite	LN/PB	Classic HL (MC), T-PLL	+	+	NA	+	+	+	+	[10]
7	55, F	2009	Composite	Cervical LN	Classic HL, PTCL	+	+	+	-	NA	+	+	[11]
8	81, F	2000	Composite	Cervical LN	Classic HL, CTCL	+	+	NA	-	+	+	+	[12]
9	88, F	1998	Composite	Inguinal LN/BM	Classic HL (MC), MF	+	†	+	†	+	+	NA	[13]
10	44, M	2017	Composite	Cervical LN	Classic HL, CTCL	+	Weak	+	+	+	+	+	[14]
11	65, M	2006	Composite	Axillary LN/liver	Classic HL, PTCL	+	-	-	-	+	+	+	[15]
12	47, M	2020	Discordant	Axillary LN	Classic HL (NS)	-	-	-	-	+	+	+	Current case
				Skin	PTCL-NOS	+	+	-	Partially positive	Weak	Weak	+	

PCR polymerase chain reaction, TCR rearrangement of T-cell receptor gene, HL Hodgkin lymphoma, RS Reed-Stenberg, LN lymph node, NS nodular sclerosis, PTCL-NOS Peripheral T-cell lymphoma, not otherwise specified, NA not applicable, MC mixed cellularity, T-PLL T-cell prolymphocytic leukemia, PB peripheral blood, BM bone marrow, CTCL cutaneous T-cell lymphoma, MF Mycosis Fungoides

†2% of T-cells were CD4/CD8 double negative in the flowcytometry

**Table 2** The current and previously reported cases with development of lymphomas following dupilumab treatment against atopic dermatitis or other chronic dermatitis

Case	Age, sex	A period of therapy for dermatitis (years)	Diagnosis before dupilumab	Time to diagnosis of lymphomas (month)	Biopsy site	Diagnosis after dupilumab	TCR	Prior or concomitant therapy for dermatitis	References
1	58, male	11	AD	1	Skin	MF	+	Topical corticosteroid, topical betametazone	[4]
2	43, male	Since childhood	AD	2	Skin	MF	NA	Intramuscular corticosteroid	[5]
3	66, female	6	CD	1.2	Skin†	MF	–	Topical and systemic steroids, phototherapy, MTX, IFN, adalimumab, mycophenolate mofeteil	[6]
4	65, female	4	AD	2.5	Skin	MF	–	Topical and systemic steroids, phototherapy, hydroxyurea	[6]
5	74, male	6	AD	24	Skin	MF	–	Topical and systemic steroids, aprelimast, pimecrolimus	[6]
6	73, male	5	CD	14	Skin†	MF	–	Topical and systemic steroids, MTX, cyclosporine, mycophenolate mofeteil, azathioprine, omalizumab	[6]
7	74, female	4	CD	12	Skin	MF	–	Topical and systemic steroids, NBUVB, MTX, mycophenolate mofeteil, secukinumab	[6]
8	44, female	9	AD	12	Skin	MF	+	topical steroids, MTX	[6]
9	27, male	7	AD	14	Skin	Suggestive of MF	NA	Topical and systemic steroids, cyclosporine	[6]
10	72, male	Over 20	AD	1.5	Skin	MF	+	Adalimumab, etanercept, MTX	[7]
11	59, female	Since childhood	AD	24	Skin†	MF	+	Mycophenolate mofeteil, cyclosporine, topical corticosteroid	[7]
12	40, female	7	AD	15	Skin	MF	–	Topical corticosteroids, clobetasol, MTX, tacrolimus ointment	[7]
13	61, male	10	CD	NA	skin†	MF	NA	Systemic predonisone	[8]



**Table 2** (continued)

Case	Age, sex	A period of therapy for dermatitis (years)	Diagnosis before dupilumab	Time to diagnosis of lymphomas (month)	Biopsy site	Diagnosis after dupilumab	TCR	Prior or concomitant therapy for dermatitis	References
14	52, male	NA	CD	3	Skin	MF	NA	Topical betametasone	[8]
15	60, male	3	CD	1.5	Skin	MF	NA	Gesekumab, topical corticosteroid, NBUVB	[8]
16	47, male	Since childhood	AD	12	axillary LN	Classic HL (NS)	–	Topical corticosteroid, antihistamine	Current case
					Skin	PTCL-NOS	+		

TCR rearrangement of T-cell receptor gene, AD atopic dermatitis, MF Mycosis Fungoides, NBUVB narrow-band ultraviolet B, MTX methotrexate, IFN interferon, CD chronic dermatitis, NA not applicable, LN lymph node, HL Hodgkin lymphoma, NS nodular sclerosis, PTCL-NOS Peripheral T-cell lymphoma, not otherwise specified

†Multiple times of biopsy were needed for diagnosis of lymphomas in these cases

have been persistent. CD30 positivity was reported to be variable among the relapsed and refractory cases after anti-CD30 antibody treatment [20]. We need to take possibility of antigen loss into consideration at histological diagnosis for recurrent or refractory cases to anti-CD30 body treatment.

In conclusion, this first case of discordant lymphomas with history of dupilumab treatment indicated that an attention is to be paid to such atopic dermatitis cases with poor response or exacerbation as to whether lymphomas associated with IL-13 and IL-4 pathway develop following blocking the pathway.

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## Declarations

**Conflict of interest** Y. M. reports lecture fees from Kyowa Kirin Co., Ltd. and Takeda Pharmaceutical Co. Ltd. S. S. reports grants, lecture fees and consultation fees from Kyowa Kirin Co., grants and lecture fees from Takeda Pharmaceutical Co. Ltd., grants from Pfizer Japan Inc., grants from Nippon Kayaku Co. Ltd., and grants and lecture fees from Sanofi K.K. M.K. reports grants and lecture fees from Kyowa Kirin Co., grants and lecture fees from Takeda Pharmaceutical Co. Ltd., grants and lecture fees from Pfizer Japan Inc., and lecture fees from Sanofi K.K. For the remaining authors none were declared.

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