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Case Report

Idiopathic multicentric Castleman disease developing after a diagnosis of sarcoidosis: A case report and literature review

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ABSTRACT

A 72-year-old woman presented with an abnormal shadow on chest radiograph. She was histologically diagnosed with sarcoidosis 20 years previously, and prednisolone was initiated 8 years previously. Computed tomography revealed centrilobular micronodules and bronchovascular bundle thickening in both lungs with multicentric lymphadenopathies; multiple pulmonary nodular lesions appeared during prednisolone tapering. Laboratory findings included polyclonal hypergammaglobulinemia and elevated interleukin-6 levels. Surgical lung biopsy revealed marked lymphoplasmacytic infiltration with lymphoid aggregates. The patient tested negative for human herpesvirus-8 and clinically diagnosed with idiopathic multicentric Castleman disease. The coexistence of sarcoidosis and Castleman disease is rare; this case improved with additional tocilizumab treatment.

1. Introduction

Castleman disease (CD) is a rare lymphoproliferative disorder, first described by Castleman et al., in 1956 [1]. It is clinically classified into unicentric CD, which involves a single enlarged lymph node, and multicentric CD (MCD), which involves multiple lymph node stations. It is histologically classified into hyaline vascular and plasma cell types with an intermediate mixed variant [2]. MCD is further classified into human herpesvirus-8 (HHV-8)-associated MCD, POEMS (polyneuropathy, organomegaly, endocrinopathy, monoclonal plasma cell disorder, skin changes)-associated MCD, and idiopathic MCD (iMCD), and has heterogeneous clinical features resulting from proinflammatory hypercytokinemia [2–4].

Sarcoidosis is an inflammatory, multisystemic disease of unknown etiology, characterized by the formation of noncaseating granulomas [5]. An increased risk of lymphoproliferative diseases, especially lymphoma, has been reported in patients with sarcoidosis [6,7]. However, an association between sarcoidosis and CD has rarely been described, and only a few reports have documented on the

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coexistence of these diseases [8–11].

Herein, we present a rare case of iMCD that developed after the diagnosis of sarcoidosis and was successfully treated with an additional use of tocilizumab, an anti-interleukin-6 (IL-6) receptor antibody.

2. Case report

A 72-year-old woman was admitted to our hospital with worsening abnormal chest shadows, assessed radiographically. The patient had bilateral hilar and mediastinal lymphadenopathy, as observed on chest computed tomography (CT) scan (Fig. 1A) and was diagnosed with sarcoidosis based on a mediastinal lymph node biopsy, revealing non-caseating epithelioid cell granulomas, 20 years ago (Fig. 1B and C). A biopsy of the facial erythema revealed skin involvement of sarcoidosis, but no ocular or cardiac lesions were noted. Prednisolone (PSL) was initiated 8 years ago owing to hypercalcemia and renal impairment, suspected as complications of sarcoidosis. At that time, CT revealed poorly defined centrilobular micronodules and bronchovascular bundle thickening in both lungs with multicentric lymphadenopathies, involving bilateral hilar, mediastinal, axillary, and inguinal regions, and splenomegaly (Fig. 2A–D), all of which improved with corticosteroid treatment.

The patient was asymptomatic, but a follow-up chest CT revealed multiple new nodular lesions in both lungs during tapering and low-dose maintenance of PSL, leading to admission (Fig. 2E). Laboratory findings on admission are summarized in Table 1. Hematological findings indicated anemia and thrombocytosis. Biochemistry revealed elevated C-reactive protein (CRP, 10 mg/dL), Krebs von den Lungen-6 (675 U/mL), immunoglobulin G (IgG, 3862 mg/dL), and IgG4 (241 mg/dL) levels. Although test results for autoantibodies such as antinuclear antibody, rheumatoid factor, and anti SS-A antibody were positive, there was no evidence of connective tissue disease. Acid-fast bacillus and fungal markers were negative. Angiotensin-converting enzyme (ACE) levels were within the normal range; however, lysozyme and soluble interleukin-2 receptor were elevated. IL-6 level was elevated at 17.9 pg/dL (<7.0). Arterial blood gas at rest showed a decrease in PaO_2 and pulmonary function tests demonstrated a decrease in carbon monoxide diffusing capacity (DLco). Bronchoscopy was proposed to rule out the differential diagnosis including malignancy, infection, or worsening of sarcoidosis, but consent was not obtained due to fear of endoscopy.

For a definitive diagnosis, video-assisted thoracoscopic surgery (VATS) of the right S8 was performed, which revealed a patchy distribution of inflammatory cell infiltration, especially plasma cells; fibrosis in the interlobular septa, bronchovascular bundles, and alveolar walls; and dense lymphoplasmacytic infiltration with lymphoid aggregates (Fig. 3A–C). Neither granulomas or malignant cells nor monoclonality was identified by immunostaining of κ and λ. Immunohistochemical staining for IgG and IgG4 showed an IgG4/IgG-positive cell ratio of approximately 30 % (Fig. 3D and E), without storiform fibrosis or obliterative phlebitis, ruling out IgG4-related disease (IgG4-RD). These histopathological findings were consistent with the plasma cell type of CD and excluded sarcoidosis, malignant disease, and IgG4-RD. Furthermore, no autoimmune disease was identified, HHV-8 was negative, and POEMS syndrome was clinicopathologically ruled out. Based on these findings, the patient was diagnosed with iMCD. Interestingly, a biopsy of an anterior thoracic skin rash 8 years ago (Fig. 4A and B) and a resection specimen of the tongue mass 2 years prior (Fig. 4C–F) had been performed, both showing lymphoplasmacytic infiltration with lymphoid follicles, suggestive of MCD-related lesions.

The clinical course of this case over 8 years following PSL initiation is shown in Fig. 5. Laboratory findings revealed that ACE level decreased after corticosteroid initiation, and high CRP levels, anemia, and hypergammaglobulinemia temporarily improved but

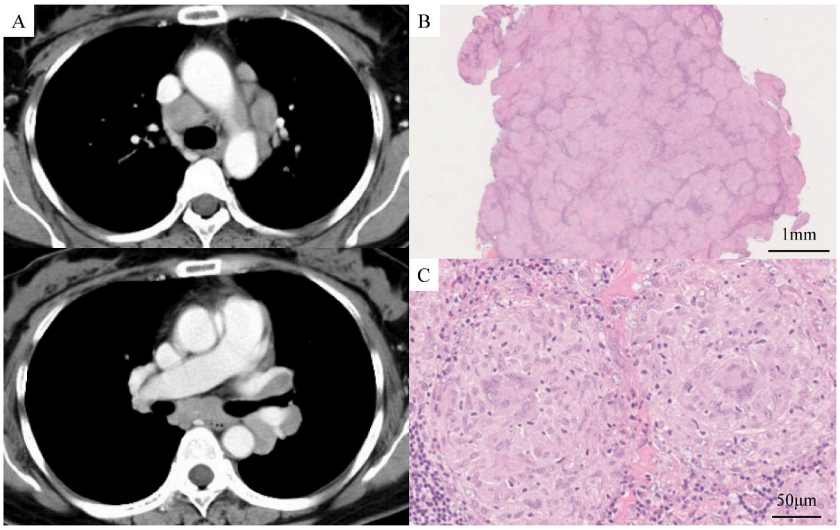

Fig. 1. Chest CT findings and histopathological findings of mediastinal lymph node biopsy 20 years ago
Chest CT shows bilateral hilar and mediastinal lymph node enlargement at the time of sarcoidosis diagnosis 20 years ago (A). Histological examination of the mediastinal lymph node biopsy shows multiple well-formed non-caseating epithelioid-cell granulomas (B, C: H&E staining).
CT: computer tomography, H&E: hematoxylin and eosin.

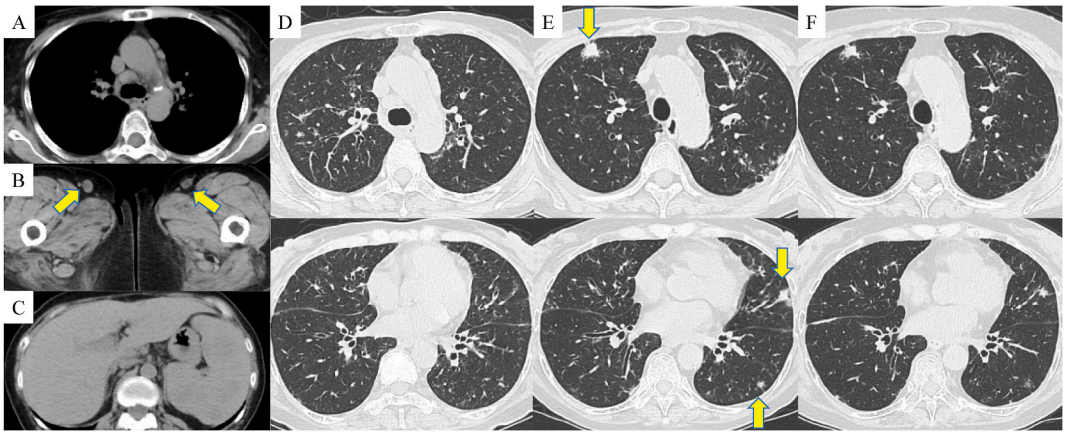

Fig. 2. CT findings during the clinical course
CT findings 8 years prior (A–D), on admission (E) and 1 year after starting tocilizumab (F). CT scan obtained at the time of hypercalcemia and renal impairment, suspected as complications of sarcoidosis 8 years prior, shows bilateral hilar, mediastinal (A), axillary, and inguinal lymphadenopathies (B: arrows), splenomegaly with hypodense nodular masses (C), and poorly defined centrilobular micronodules and bronchovascular bundle thickening in both lungs (D). These findings resolved with corticosteroid treatment. Chest CT scan obtained at the time of surgical lung biopsy shows that new multiple nodular lesions (arrows) emerged during tapering and low-dose maintenance of corticosteroid (E). Approximately 1 year after starting tocilizumab, these pulmonary lesions were reduced (F).
CT: computed tomography.

Table 1
Laboratory findings at the time of admission.

Hematology			LD	93	U/L	T-spot	(−)	
WBC	6480	/μL	ALP	81	U/L	β-D-glucan	5.9	pg/mL
Neut	58.2	%	γGTP	12	U/L	Aspergillus Ag	0.3	
Lym	25.9	%	HbA1c	6.5	%	Cryptococcus Ag	(−)	
Eos	2.3	%	KL-6	675	U/mL	CEA	2.3	ng/mL
Hb	8.2	g/dL	SP-D	55.9	ng/mL	CYFRA	2.4	ng/mL
Plt	42.2	$\times 10^4$/μL				proGRP	77.1	pg/mL
			Serology			sIL-2R	1020	U/mL
Chemistry			CRP	10.4	mg/dL			
TP	9.0	g/dL	IgG	3862	mg/dL	Arterial blood gas (room air)		
Alb	2.5	g/dL	IgG4	241	mg/dL	pH	7.474	
BUN	19	mg/dL	IgA	365	mg/dL	$PaCO_2$	38.7	Torr
Cre	0.82	mg/dL	IgM	224	mg/dL	PaO_2	60.1	Torr
Na	141	mEq/L	IgE	483	IU/mL			
K	3.7	mEq/L	IL-6	17.9	pg/mL	Pulmonary function tests		
Cl	105	mEq/L	ACE	20.0	U/L	FVC	2.14	L
Ca	8.6	mg/dL	Lysozyme	15.5	μg/mL	%FVC	98.2	%
P	3.1	mg/dL	ANA	× 40		FEV_1	1.87	L
AST	12	U/L	RF	22	IU/mL	$\%FEV_1$	109.0	%
ALT	5	U/L	Anti-SS-A Ab	13.1	U/mL	FEV_1/FVC	78.0	%
T-Bil	0.3	mg/dL	Anti-SS-B Ab	1.5	U/mL	%DLco	56.8	%

Ab, antibody; Ag, antigen; ACE, angiotensin-converting enzyme; Alb, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; ANA, antinuclear antibody; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CEA, carcinoembryonic antigen; Cre, creatinine; CYFRA, cytokeratin 19 fragment; CRP, C-reactive protein; DLco, diffusing capacity for carbon monoxide; Eos, eosinophil; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; γGTP, gamma-glutamyl transpeptidase; IL-6, interleukin-6; KL-6, Krebs von den Lungen-6; Hb, hemoglobin; Ig, immunoglobulin; LD, lactate dehydrogenase; Lym, lymphocytes; Neut, neutrophil; $PaCO_2$, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen; Plt, platelets; proGRP, pro-gastrin releasing peptide; RF, rheumatoid factor; sIL-2R, soluble interleukin-2 receptor; SP-D, surfactant protein-D; T-Bil, total bilirubin; WBC, white blood cells.

became apparent during PSL tapering. After diagnosing the case as iMCD, tocilizumab was initiated in addition to the maintenance of PSL at 5 mg/day based on informed consent. Following tocilizumab initiation, laboratory findings showed that CRP normalized and anemia and hypergammaglobulinemia tended to improve (Fig. 5). In addition, chest CT at 1 year found that pulmonary lesions, including multiple nodules, were reduced (Fig. 2F). Approximately 1 year after starting tocilizumab, no worsening of sarcoidosis or iMCD had been reported.

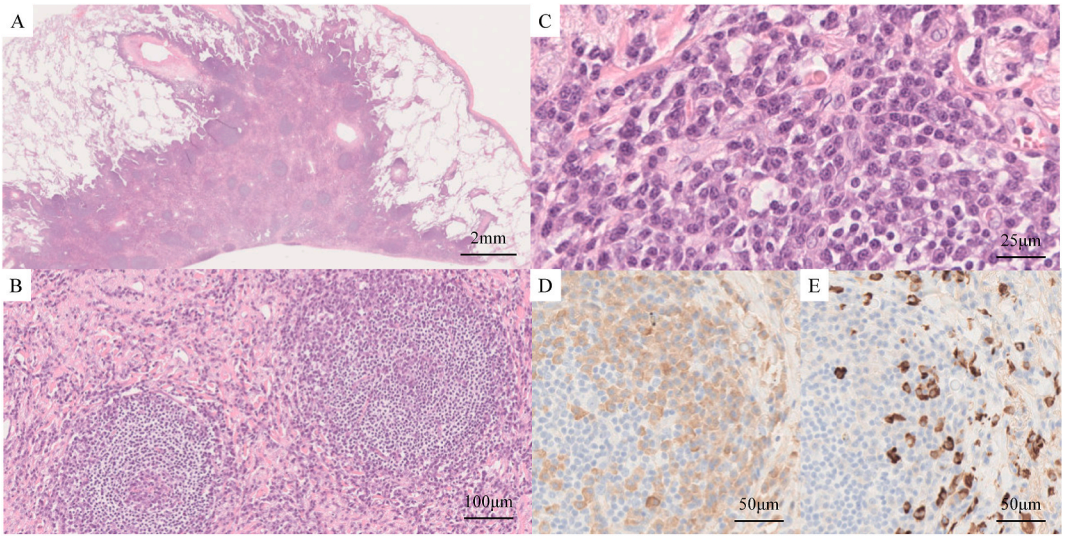

Fig. 3. Histopathological findings on surgical lung biopsy
The VATS biopsy specimen obtained from the right S^8 shows a patchy distribution of inflammatory cell infiltration and fibrosis in the interlobular septa, bronchovascular bundles, and alveolar walls (A: H&E staining). There is a dense lymphoplasmacytic infiltration with lymphoid aggregates (B: H&E staining), and plasma cell infiltration is prominent (C: H&E staining). Immunohistochemical staining for IgG (D) and IgG4 (E) shows an IgG4/IgG-positive cell ratio of approximately 30 %.
H&E: hematoxylin and eosin, IgG: immunoglobulin G, VATS: video-assisted thoracoscopic surgery.

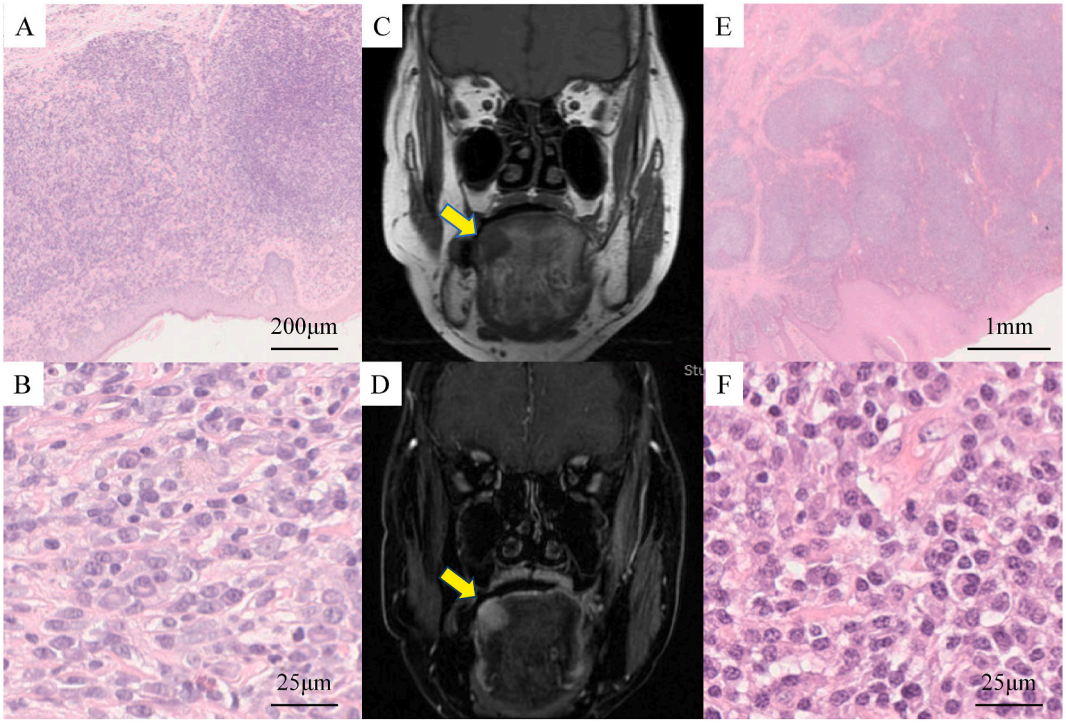

Fig. 4. Histopathological findings of specimens obtained from skin rash and tongue mass and MRI findings of tongue lesion
Skin biopsy of the anterior thoracic rash (8 years ago) shows inflammatory cell infiltration with predominantly plasma cells and proliferation of lymphoid follicles beneath the epithelium (A, B: H&E staining). A tongue lesion with induration was noted 2 years ago, and MRI revealed an approximately 2 cm tongue mass at the right edge of the tongue (arrows), showing low signal intensity on a T1-weighted image (C) and low signal intensity on a T2-weighted image (D). The lesion was resected to rule out malignancy. A resected specimen from the tongue mass shows marked lymphoplasmacytic infiltration with lymph follicles (E, F: H&E staining).
H&E: hematoxylin and eosin staining, MRI: magnetic resonance imaging.

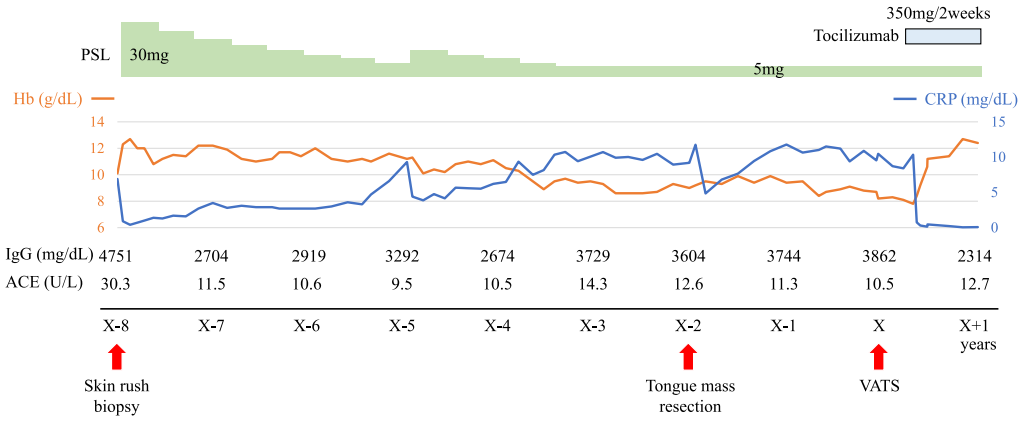

Fig. 5. Clinical course after the corticosteroid therapy
ACE, angiotensin-converting enzyme; CRP, C-reactive protein; Hb, hemoglobin; IgG, immunoglobulin G; PSL, prednisolone; VATS, video-assisted thoracoscopic surgery.

3. Discussion

In the present report, we describe a rare case of MCD that developed following a diagnosis of histologically confirmed sarcoidosis. Although hypercalcemia and renal dysfunction 8 years ago were suspected to be complications of sarcoidosis, this patient also presented with multicentric lymphadenopathy, high CRP levels, anemia, and polyclonal hypergammaglobulinemia, suggesting that MCD may have been present concurrently. Although splenomegaly can result from either sarcoidosis or iMCD [3,5], multiple hypodense nodular masses in the spleen suggested sarcoidosis. Notably, new pulmonary lesions appeared during tapering and low-dose maintenance of PSL, and VATS biopsy findings confirmed plasma cell-type CD, with no evidence of sarcoidosis or malignancy. Superficial lymphadenopathies had resolved after corticosteroid treatment, preventing mediastinal lymph node biopsy due to adhesions resulting from a previous mediastinoscopic lymph node biopsy. However, based on the clinicopathological findings and diagnosis by exclusion, the patient was finally diagnosed with iMCD. The pulmonary lesions gradually improved after the initiation of tocilizumab. These findings suggest that sarcoidosis and iMCD can coexist and IL-6 inhibition serves as a valuable treatment option.

The coexistence of sarcoidosis and CD has rarely been reported, and to the best of our knowledge, only five cases have been documented, as summarized in Table 2 [8–11]. These cases were relatively common in middle-aged women, and sarcoidosis and MCD occur simultaneously or one precedes the other [9–11]. In the reports from Japan [8,10], MCD often developed several years after the diagnosis of sarcoidosis. In the present case, iMCD was histologically confirmed 20 years after the diagnosis of sarcoidosis but could have been present for at least 12 years. The diagnosis of CD is typically based on lymph node and/or surgical lung biopsy [8–11], and the plasma cell type being most common [9,11]. In cases without superficial lymphadenopathy, surgical lung biopsy may be useful for diagnosing MCD [11], although invasive procedure such as surgical lung biopsy should be considered only when other less invasive procedures are inconclusive or they are not available at respective center. In this case, endobronchial ultrasound-guided transbronchial needle aspiration or biopsy was a potentially useful diagnostic tool, but unfortunately, consent for bronchoscopy was not obtained. All reported cases have shown improved outcomes following treatment with PSL and anti-IL-6 inhibitors [8–11]. Similarly, tocilizumab was effective in treating iMCD in the present case.

Table 2
Clinical characteristics of cases with sarcoidosis and Castleman disease.

Author, Year	Age[a]/sex	Diagnosis, duration	Biopsy	Pathology of CD	Treatment	Outcome
Rice et al., 2011 (8)	37/F	Sar, UCD, Concomitant	Sar: LN, lung CD: LN	HV type	PSL, MTX	Improvement
Awano et al., 2012 (9)	60/F	Sar→MCD, 17 years	Sar: Skin, breast CD: LN	Mixed type	Sar: none CD: PSL, tocilizumab	Improvement
	41/M	Sar→MCD, 3 years	N/A	PC type	Sar: none CD: PSL	Improvement
Mohammed et al., 2015 (10)	76/M	MCD→Sar, 5 months	CD: Lung Sar: LN	N/A	CD: situximab Sar: PSL, MTX	Improvement
Sawata et al., 2016 (11)	60/F	Sar→MCD, 8 years	Sar: Lung CD: Lung	PC type	Sar: none CD: PSL	Improvement
The present case	52/F	Sar→MCD, 20 years	Sar: LN, skin CD: Lung, skin, tongue	PC type	Sar: PSL CD: tocilizumab	Improvement

CD, Castleman disease; F, female; HV, hyaline vascular; LN, lymph node; M, male; MCD, multicentric Castleman disease; MTX, methotrexate; N/A, not available; PC, plasma cell; PSL, prednisolone; Sar, Sarcoidosis; UCD, unicentric Castleman disease.
[a] Age at diagnosis of the preceding disease.

The mechanisms underlying the development of sarcoidosis and MCD are unclear; however, IL-6, a critical disease driver in MCD [2], has been reported to be elevated in serum and bronchoalveolar lavage fluid of patients with sarcoidosis [12,13]. In particular, IL-6 level is higher in patients with sarcoidosis who require corticosteroid therapy or have progressive disease than those with stable or improved disease, suggesting its correlation with disease activity [12]. In addition, sarcoidosis is associated with lymphoproliferative disorders, especially lymphoma [6,7], and patients with MCD have also been reported to have a high incidence of lymphoma [3,14]. These findings suggest a common pathogenic mechanism for sarcoidosis and MCD.

In the present case, findings from previous biopsy specimens of skin rash (8 years ago) and tongue mass (2 years ago) suggested cutaneous and tongue lesions associated with iMCD. Cutaneous plasmacytosis is a rare manifestation of iMCD that can precede any other indications of the disease [15,16]. Tongue involvement in CD is extremely rare, with only one case of HHV-8-associated MCD reported to date in an HIV-positive patient [17].

Recently, iMCD has been further subclassified into iMCD-thrombocytopenia, ascites, reticulin fibrosis, renal dysfunction, organomegaly (iMCD-TAFRO) and iMCD-not otherwise specified (iMCD-NOS) [2,4]. The clinical features of the present case were distinct from those of iMCD-TAFRO and included polyclonal hypergammaglobulinemia, thrombocytosis, and histopathological findings of plasmacytic CD, which are suggestive of iMCD-NOS, particularly the idiopathic plasmacytic lymphadenopathy type [4,18].

There is no established evidence for concomitant treatment of sarcoidosis and CD. Tocilizumab, which targets the IL-6 receptor, was approved in Japan in 2005 and has been used worldwide to treat iMCD [3,19]. However, the potential use of IL-6R inhibition for sarcoidosis management remains unclear. The use of tocilizumab, as a novel corticosteroid-sparing therapy, was reported in a case series of four patients with sarcoidosis [20]; however, tocilizumab-induced sarcoidosis has been reported as an adverse event during tocilizumab therapy [21]. Mohammed et al. reported a case in which siltuximab treatment for MCD resulted in a mixed response, with improvement in lung lesions and worsening of mediastinal lesions, confirming the development of sarcoidosis on mediastinal lymph node biopsy [10]. Although additional treatment with tocilizumab for iMCD produced a favorable response in the present case, careful follow-up is needed to monitor the worsening of sarcoidosis.

In conclusion, we reported a case of iMCD that developed after a diagnosis of sarcoidosis. The addition of tocilizumab to the treatment regimen was effective in this case. Although the coexistence of sarcoidosis and CD is rare, it is an important condition to consider.

CRediT authorship contribution statement

Takumi Muramatsu: Writing – review & editing, Writing – original draft, Data curation. **Masato Kono:** Writing – review & editing, Writing – original draft, Data curation, Conceptualization. **Masaki Ishige:** Writing – review & editing, Data curation. **Takahiko Saito:** Writing – review & editing, Data curation. **Misato Higasa:** Writing – review & editing, Data curation. **Fumiya Nihashi:** Writing – review & editing, Data curation. **Yuya Aono:** Writing – review & editing, Data curation. **Mineo Katsumata:** Writing – review & editing, Data curation. **Hideki Miwa:** Writing – review & editing, Data curation. **Yoshihiro Miki:** Writing – review & editing, Data curation. **Taro Misaki:** Writing – review & editing, Supervision, Data curation. **Yoshiro Otsuki:** Writing – review & editing, Supervision. **Dai Hashimoto:** Writing – review & editing, Supervision, Data curation. **Noriyuki Enomoto:** Writing – review & editing, Supervision. **Takafumi Suda:** Writing – review & editing, Supervision. **Hidenori Nakamura:** Writing – review & editing, Supervision.

Consent for publication

Informed consent was obtained from the patient.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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