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【 CASE REPORT 】

Lupus Aortitis Successfully Treated with Moderate-dose Glucocorticoids: A Case Report and Review of the Literature

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Abstract:
Lupus aortitis is a rare and potentially life-threatening disorder. Previous studies have reported the utility of high-dose systemic glucocorticoids or surgery as the treatment, although there have been no related controlled trials. We herein report a 49-year-old woman with a 35-year history of systemic lupus erythematosus who was diagnosed with aortitis. Her symptoms and laboratory and imaging abnormalities rapidly resolved upon the administration of moderate-dose glucocorticoids. We subsequently performed a literature review of similar cases to identify the appropriate treatment and discuss these cases. A study of further cases will be needed to identify the characteristics of patients who would benefit from moderate-dose glucocorticoid therapy.

Key words: systemic lupus erythematosus, lupus aortitis, moderate-dose glucocorticoid therapy

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Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disorder associated with multisystem organ damage mediated by autoantibodies and immune complexes. Aortitis is an uncommon complication of SLE (1-27). Aortic dissection and aortic aneurysmal rupture are potentially fatal complications of lupus-associated aortitis (lupus aortitis) (7-9, 12-17, 22, 24).

Many previous reports on lupus aortitis have stated the need for therapy with high-dose systemic glucocorticoids or surgery (5-10, 12-15, 17-27). However, glucocorticoid therapy is associated with both accelerated atherosclerosis, which causes cardiovascular events, and aortic aneurysmal enlargement (28-31). It is therefore important to limit the exposure to glucocorticoids, particularly in patients with aortic aneurysms. However, the appropriate amount and duration of glucocorticoid therapy for lupus aortitis remains unclear because of a lack of controlled trials.

We herein report a case of lupus aortitis that was successfully treated with moderate-dose glucocorticoids. The current report is significant because there are no previous reports in which remission was successfully induced by conservative therapy with moderate doses of glucocorticoids.

Case Report

A 49-year-old woman was admitted with a 1-week history of bilateral shoulder pain that migrated to the precordium and a 2-day history of a fever and dyspnea. Although the fever was resolved with oral loxoprofen, her chest pain remained.

She had been diagnosed with SLE at 14 years of age, after she presented with facial erythema, photosensitivity, and hair loss as well as laboratory results of positive antinuclear antibodies, positive anti-double stranded deoxyribonucleic acid (anti-dsDNA) antibodies, and hypocomplementemia. She subsequently developed both pericarditis and pleurisy several times. These conditions were resolved with prednisolone (PSL) at a dose of about 0.5 mg/kg/day. During all of her previous episodes, she had experienced chest pain that was exacerbated by movement and breathing, along with a fever, and elevated anti-dsDNA antibodies and elevated C-

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Table 1. Laboratory Parameters on Admission.

Investigations (unit)	
Hemoglobin (g/dL)	9.6
Mean corpuscular volume (fL)	77.4
Mean corpuscular hemoglobin consentration (g/dL)	30.5
Reticulocyte count (%)	0.9
Iron (μg/dL)	19
Ferritin (ng/mL)	49
Transferrin (mg/dL)	221
Coombs test	negative
Total leukocyte count (/μL)	5,860
Lymphocyte count (/μL)	1,030
Platelet count (×10,000/μL)	36.9
CRP (mg/dL)	9.4
Erythrocyte sedimentation rate (mm/hour)	89
Creatinine (mg/dL)	0.6
CH50 (U/mL)	48.3
C3 (mg/dL)	136
C4 (mg/dL)	27.8
Antinuclear antibodies (times)	640 (Homogene, Speckled)
Anti-double stranded deoxyribonucleic acid antibodies (IU/mL)	13
Anti-Sm antibodies (U/mL)	negative
Anti-U1 ribonucleoprotein antibodies (U/mL)	negative
Anti-Ro/SSA antibodies (U/mL)	>500
Anti-La/SSB antibodies (U/mL)	>500

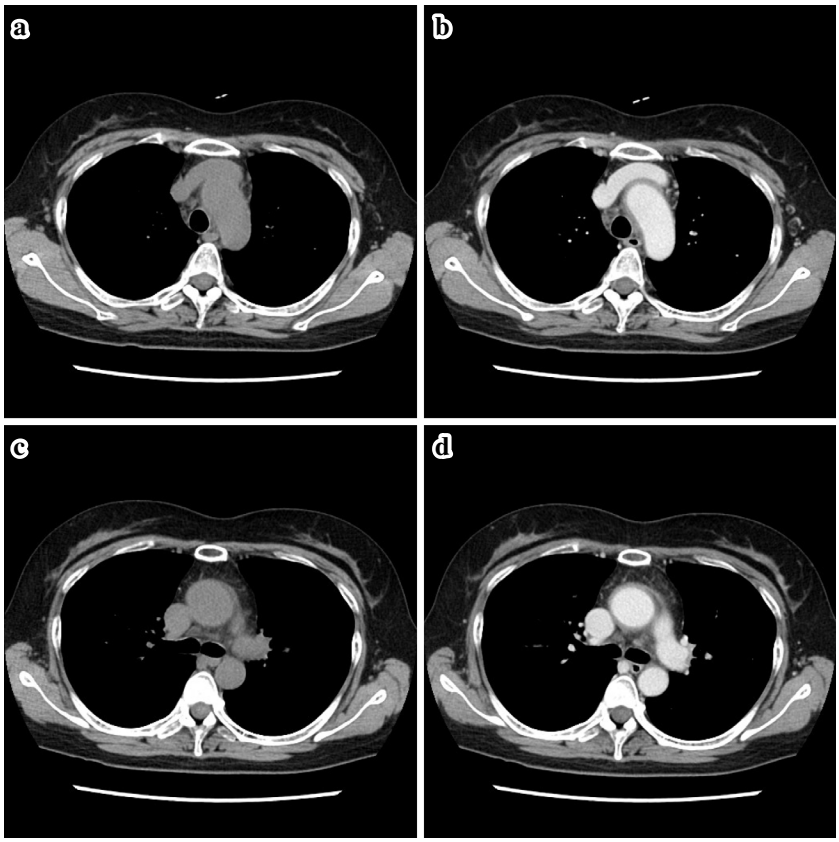

Figure 1. Contrast-enhanced CT on admission showed abnormal thickening and enhancement of the aortic wall from the ascending aorta to the arch.

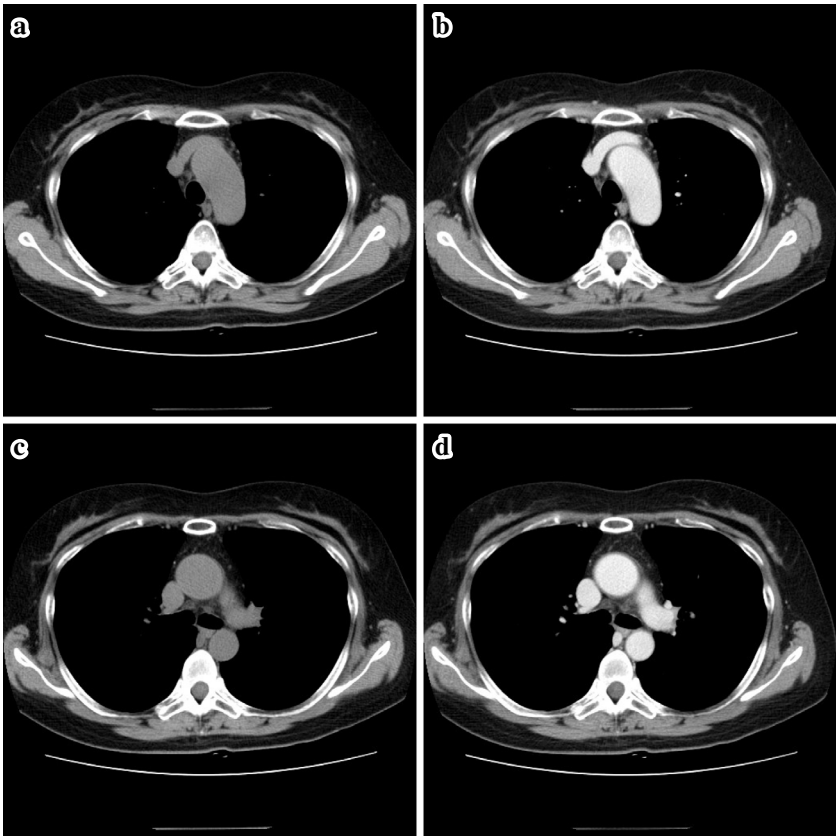

Figure 2. Contrast-enhanced CT performed two weeks after the PSL dose was increased revealed disappearance of the aortic wall thickening and periaortic soft tissue inflammation.

reactive protein (CRP) levels were seen. In addition, the episodes of pleurisy were accompanied by pleural effusion. PSL therapy was gradually reduced to 8 mg/day orally, and she visited the hospital regularly for observation while maintaining this dose for 29 months. She had also been diagnosed with Sjögren's syndrome based on her dry mouth and positive findings for anti-Ro/SSA and anti-La/SSB antibodies.

She was completely alert on the day of admission, and her vital signs were as follows: blood pressure, 114/75 mmHg; pulse rate, 82 beats per minute; body temperature, 36.0℃; respiratory rate, 16 breaths per minute; and peripheral capillary oxygen saturation level on ambient air (SpO_2) of 98%. A physical examination showed a height of 156 cm and weight of 56.6 kg. Cardiovascular, respiratory, and abdominal examinations were normal, although she complained of tenderness over the sternum.

A laboratory examination revealed a white blood cell count of 5,860/μL, hemoglobin of 9.6 g/dL, platelet count of 369,000/mm^3, CRP of 9.4 mg/dL, erythrocyte sedimentation rate of 89 mm/h, and anti-dsDNA antibodies of 13 U/mL (Table 1). She had presented with iron deficiency anemia for five years. Anti-dsDNA antibodies had been positive for 13 years and increased to more than 50 U/mL when she developed both pericarditis and pleurisy. Complement levels, creatinine, and a urinalysis showed no abnormalities.

Plain chest X-ray, an electrocardiogram, and transthoracic echocardiography findings were all normal. However, contrast-enhanced CT revealed thickening of the aortic wall from the ascending aorta to the arch, along with periaortic soft tissue inflammation (Fig. 1).

Additional tests of blood culture, interferon-γ release assay, β-D-glucan, HBs-antigen/HBs-antibody, HCV-antibody, IgG-4, myeloperoxidase-anti-neutrophil cytoplasmic antibody (ANCA), and Proteinase 3-ANCA after admission were negative. Her human leukocyte antigen was A2, B51, B62, although she had no history of oral or genital ulcers, ocular lesions, or cutaneous lesions, such as pathergy reactions, erythema nodosum and pseudofolliculitis suggestive of Behcet's disease. She did not present with bloody diarrhea or abdominal pain. Anti-β2-glycoprotein I antibody, lupus anticoagulant, and anti-cardiolipin antibody were positive. She had no history of thrombosis and had had two pregnancies and deliveries. Brain magnetic resonance imaging revealed no ischemic changes. Antitreponemal antibody was negative. Based on these findings, we diagnosed her with lupus aortitis.

Since she strongly desired the same moderate-dose PSL therapy as before, and with the intention of minimizing the adverse effects of glucocorticoids, we started PSL at a dose of 30 mg/day (0.5 mg/kg/day). She did not approve of our suggestion that she should take hydroxychloroquine (HCQ) as standard therapy for SLE or another immunosuppressant as a glucocorticoid-sparing drug because she was afraid of developing an allergy. As a result, her chest pain resolved, and her inflammatory marker levels and anti-dsDNA anti-

Table 2. **Literature Review of Cases of Lupus Aortitis.**

Reference	Age	Sex	Symptoms	Diagnosis	Site	Aneurysm	Dissection	Prior treatment	Treatment for aortitis at admission	Outcome	Pathological findings
1	63	F	Extremity claudication	Symptoms, examination	Carotid artery, subclavian artery, abdominal aorta	Unknown	Unknown	Unknown	Corticosteroid	Poor	Unknown
2	46	M	Dyspnea	Autopsy	A. valve-Aorta	(-)	(-)	PSL 10-30 mg	Corticosteroid	Death	Obliterative endarteritis of the vasa vasorum and perivascular lymphocytic infiltration in the adventitia and outer media
3	34	F	Dyspnea	Cardiac catheterization	A. valve-Ascending	(-)	(-)	PSL 60→ 10 mg	Continuous PSL 10 mg, diuretic	Survival for >1 year	Unknown
4	59	F	Poor pulse on palpation	Angiography	Arch-Abdominal	(-)	(-)	Unknown	Unknown	Unknown	Unknown
5	29	F	Poor pulse on palpation	Angiography	Arch-Abdominal	(-)	(-)	None	PSL 60 mg	Recovery	Unknown
6	19	F	AR, heart failure	Autopsy	A. valve-Ascending	(-)	(-)	Low-dose PSL	PSL 40 mg→ 5 mg, surgery	Death (heart failure)	Perivascular lymphoplasmacytic infiltration with obliterative endarteritis of the vasa vasorum in the adventitia and media, disruption of the elastic lamina with neovascularization and fibrosis in the media, irregular thickening of the intima and cholesterol deposition
7	56	M	None	Resected specimen	Abdominal	(+)	(-)	PSL	Surgery	Recovery	Obliterative endarteritis, fibrinoid necrosis of the vasa vasorum and lymphocyte infiltration around the vasa vasorum in the adventitia, disruption of the elastic lamina in the media, calcification of the intima and cholesterol deposition
8	30	F	None	CT, resected specimen	Ascending	(+)	(-)	PSL 5 mg	Surgery	Death (intraabdominal hemorrhage)	Obliterative endarteritis of the vasa vasorum and perivascular lymphocytic infiltration in all layers, disruption of the elastic lamina and hyperplasia of collagen fiber in the media, plaque on the intima
9	31	F	Chest pain radiating to back	Autopsy	A. valve-Ascending	(-)	(+)	PSL 5-30 mg, AZA, HCQ	mPSL planned	Death (tamponade)	Obliterative endarteritis and fibrinoid necrosis of the vasa vasorum and lymphocytic infiltration around infarction sites in the adventitia and media, disruption of the elastic lamina in the media, plaque formation on the intima
10	6	F	Poor pulse on palpation	Angiography	Arch-Thigh	(-)	(-)	PSL	PSL 2 mg/kg, surgery	Recovery	Unknown
11	27	F	Leg coldness/pain	Autopsy	Arch	(-)	(-)	PSL, AZA	PCI, urokinase	Death	Disruption of the media due to lymphoplasmacytic infiltration, immune complex deposition by IgG, C3 and fibrinogen in the media, thrombus adhesion in the lumen without obvious arteriosclerosis in the intima
12	40	M	Chest pain, dyspnea	TEE, resected specimen	A. valve-Arch	(+)	(+)	Corticosteroid, AZA	Surgery	Recovery	Chronic nonspecific perivasculitis of the adventitia, multiple small necrosis in the media
13	47	F	Back pain	MRI, resected specimen	Ascending-Arch	(+)	(-)	Unknown	Surgery	Recovery	Fibrosis and lymphocytic infiltration in the adventitia, extensive necrosis of the media and surrounding granulomatous tissue, worm-eaten disruption in the media, plaque formation on the intima
14	37	M	Back pain	Resected specimen	Abdominal	(+)	(-)	mPSL pulse → PSL tapered to 10 mg, IVCY 1 g/m^2 12 times	Surgery	Recovery	Obliterative endarteritis of the vasa vasorum in the adventitia, disruption of medial and adventitial layers with destroyed elastic laminae
15	36	M	None	Autopsy	Ascending	(-)	(+)	PSL 30 mg	mPSL, hydrocortisone	Death (tamponade)	Obliterative endarteritis of the vesa vasorum in the adventitia, fibrinoid necrotizing vasculitis and microscopic aneurysms in the kidneys, pancreas, spleen, and pleura
16	44	F	Weight loss, fatigue	Biopsy, resected specimen		(-)	(-)	Unknown	Unknown	Unknown	Vasculitis of the aorta, internal thoracic artery, and saphenous vein
17	34	F	Pericarditis	Resected specimen	Ascending	(+)	(-)	Unknown	Unknown	Unknown	Fibrosis and neovascularization in the adventitia

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Table 2. **Literature Review of Cases of Lupus Aortitis. (Continued)**

Reference	Age	Sex	Symptoms	Diagnosis	Site	Aneurysm	Dissection	Prior treatment	Treatment for aortitis at admission	Outcome	Pathological findings
18	35	F	None	CT, resected specimen	Descending	(+)	(-)	PSL	PSL, surgery	Recovery	Obliterative endarteritis of the vasa vasorum in the adventitia, worm-eaten disruption of the elastic lamina in the media, perivascular lymphoplasmacytic infiltration in the adventitia and media, calcifications and atheroma within the thickened intima
19	36	F	Left hemiplegia	MRA, angiography	Internal carotid artery, renal artery	(-)	(-)	None	PSL 75 mg, CY 2 mg/kg	Recovery	Unknown
20	30	M	Abdominal pain, vomiting	CT, intraoperative findings	Arch	(-)	(-)	PSL 60 mg	PSL 60 mg, MMF, Surgery	Recovery	Small-vessel vasculitis accompanying intravascular thrombi in the pericardial vasculature
21	32	F	None	Autopsy		(-)	(-)	mPSL pulse, PSL, AZA, IVCY	mPSL, HCQ	Death	Systemic small-vessel vasculitis including the vasa vasorum
22	57	M	Fever, chest pain	PET	Thoracic	(-)	(-)	mPSL 32 mg → Discontinued	mPSL 32 mg	Recovery	Unknown
23	23	F	Fever, pleural pain	CT, MRI, resected specimen	Ascending	(+)	(-)	None	mPSL pulse, MTX, high-dose PSL, surgery	Recovery	Obliterative endarteritis in the adventitia, patchy necrosis in the media
24	28	M	Abdominal pain, nausea	Contrastenhanced CT	Thoracic-Abdominal	(-)	(-)	Warfarin	PSL pulse, PSL 60 mg, IVCY, MMF	Recovery	Unknown
25	30	F	Chest pain	Contrastenhanced CT	Ascendingexternal Iliac	(-)	(+)	Betamethasone 1.5 mg	Betamethasone 3 mg, Surgery	Recovery	Dissection of the elastic media, obliterative endarteritis of the vasa vasorum
26	23	F	Fever, dyspnea, chest pain	CT, MRI, resected specimen	Ascending	(-)	(-)	None	mPSL pulse, PSL 1 mg/kg/day	Death (graft infection)	Diffuse lymphocytic infiltration, disruption of the elastic lamina and necrosis of the media
27	17	F	Generalized edema	Autopsy		(-)	(-)	None	mPSL pulse	Death	Systemic polyangiitis, lymphocytic infiltration of all layers of the aorta
28	21	F	Dyspnea	PET, resected specimen	Ascending-Arch	(-)	(-)	None	High-dose PSL	Recovery	Obliterative endarteritis and perivascular lymphocytic infiltration in the adventitia and media, necrosis with neovascularization of the media
29	49	F	Fever, dyspnea, migratory chest pain	Contrastenhanced CT	Ascending-Arch	(-)	(-)	PSL 8 mg	PSL 30 mg (0.5 mg/kg)	Recovery	Unknown

AR: aortic regurgitation, A.valve: aortic valve, AZA: azathioprine, CT: computed tomography, CY: cyclophosphamide, HCQ: hydroxychloroquine, IVCY: Intravenous cyclophosphamide, MMF: mycophenolate mofetil, mPSL: methylprednisolone, MRA: magnetic resonance angiography, MRI: magnetic resonance imaging, MTX: methotrexate , PCI: percutaneous coronary intervention, PET: positron emission tomography, PSL: prednisolone, TEE: transesophageal echocardiography

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bodies became negative after two weeks. Contrast-enhanced CT performed two weeks after the increased PSL dose revealed disappearance of the aortic wall thickening and periaortic soft tissue inflammation (Fig. 2). She was discharged on day 21.

PSL was continued at the initial dose for two weeks. Subsequently, the dose was gradually reduced by 5 mg every 2 weeks to 15 mg, and then by 2.5 mg every 4 weeks to 10 mg. As of 22 months after the symptom onset, there has been no recurrence with a dose of 7.5 mg of PSL.

Discussion

We described the first case of lupus aortitis that responded to conservative treatment with moderate doses of PSL. There has been no textbook or systematic review describing the characteristics of lupus aortitis or its treatment strategy. There have been several cohort studies of vasculitis in SLE (32, 33), and aortitis was not mentioned in any of those reports.

In February 2020, we searched PubMed, Google Scholar, and Google for previous reports on lupus aortitis using the terms "SLE, aortitis" and "lupus, aortitis" in English and "SLE, aortitis" in Japanese. We identified 28 such cases of lupus aortitis in review papers and their cited references in English or Japanese (1-27) (Table 2). Since nine of the cases were fatal, lupus aortitis was thought to be a serious condition. The causes of death in many of these cases were attributed to complications from aortic dissection due to active aortitis or to postoperative complications (8, 9, 15, 25). Among the patients who recovered, surgery was performed for aortic dissection or aortic aneurysm, although most reports concerning surgical aortic repair did not mention the induction dose of corticosteroids (7, 8, 12-14, 17). High-dose glucocorticoids have been recommended as the initial treatment for Takayasu's arteritis or giant cell arteritis, which are major types of aortitis (34, 35). However, fatal cases of lupus aortitis without aortic dissection or aortic aneurysm despite high-dose glucocorticoid administration have been reported (25, 26). Since heterogeneous outcomes have been reported and there have been no controlled trials, the need for high-dose glucocorticoids as the initial treatment for lupus aortitis is unclear.

However, it has been reported that glucocorticoid administration itself can induce atherosclerotic changes and contribute to the fragility of the aortic tunica media, which might induce aortic aneurysmal enlargement (12, 13, 15, 28-31). Therefore, if possible, medical intervention with moderate-dose glucocorticoids seems beneficial for limiting the amount of glucocorticoids administered. It may thus be reasonable to consider initial treatment with moderate-dose glucocorticoids for lupus aortitis when there is no aortic dissection or aortic aneurysm formation at the initial evaluation and close follow-up is possible, or when there are additional factors that are relative contraindications to high-dose glucocorticoids.

Our patient showed no serious complications, such as aortic dissection or aortic aneurysm formation. After explaining the risks associated with insufficient treatment to this patient, moderate-dose glucocorticoid therapy was started, which successfully induced remission. The best immunosuppressant for lupus aortitis is unclear (18, 19, 22, 23). If the patient's aortitis had not responded to the initial treatment, we would have increased the dose of PSL and persuaded her to take an immunosuppressant, such as cyclophosphamide or mycophenolate mofetil, while sharing information about the adverse events associated with the immunosuppressant. We intend to add HCQ if she agrees to take it, as HCQ is recommended for all SLE patients as the standard therapy (36).

There have been no previous reports of successful remission of lupus aortitis with moderate-dose glucocorticoids. It will therefore be necessary to examine more cases in the future to identify the characteristics of patients who are likely candidates for successful treatment with moderate doses of glucocorticoids.

The authors state that they have no Conflict of Interest (COI).

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