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CASE REPORT
Cardiac imaging

Granulocyte colony stimulating factor-associated aortitis evaluated via multiple imaging modalities including vascular echography: a case report

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Background Granulocyte colony stimulating factor (G-CSF) preparations are used for patients with granulocytopenia, especially to prevent febrile neutropenia. Arteritis has been recognized as a side effect of G-CSF treatment; however, there are no clear diagnostic criteria or treatment guidelines because not enough cases have been reported. Present case showed one of the diagnostic and treatment selection methods via multiple imaging modality including vascular echography.

Case summary A 52-year-old woman underwent chemotherapy for ovarian cancer and received G-CSF because of myelosuppression. The patient experienced high and remittent fever that persisted during treatment using antibiotics and acetaminophen. Enhanced computed tomography revealed thickening of the tissue around the aortic arch and abdominal aorta. Echography of the abdominal aorta revealed thickening of the wall and a hypoechoic region around the aorta. Gadolinium-enhanced magnetic resonance imaging and ^{18}F-fludeoxyglucose positron emission tomography also revealed that the inflammation was localized to the lesion. A suspicion of G-CSF-associated aortitis was based on the patient's history and the exclusion of other diseases that might have caused the aortitis. Her condition rapidly improved after starting corticosteroid treatment.

Discussion The differential diagnosis in similar cases should consider immune diseases that cause large-vessel arteritis (Takayasu arteritis, giant cell arteritis, and another vasculitis), infection, drug-induced disease, and immunoglobulin G4-related disease. The use of different imaging modalities, including vascular echography, helped guide the diagnosis and follow-up. It is necessary to evaluate the patient's general condition before the selection of treatments.

Keywords Granulocyte colony stimulating factor (G-CSF) • Aortitis • Arteritis • Vascular echography • Onco-cardiology • Case report

Learning points

- In patients with persistent fever and a history of granulocyte colony stimulating factor administration, aortitis should be considered in the differential diagnosis.
- The arteritis localization and activity should be evaluated using multiple modalities (e.g. vascular echography, computed tomography, and magnetic resonance imaging) including neck to the pelvis to guide treatment.
- The treatment including the necessity of corticosteroid therapy should be selected based on the general condition of the patients, considering that the patients are treated for cancer.

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Introduction

Granulocyte colony-stimulating factor (G-CSF) preparations are used for chemotherapy-related granulocytopenia, especially to prevent febrile neutropenia. The G-CSF preparations bind to G-CSF receptors that are present on neutrophil progenitor cells in the bone marrow, which promote their differentiation into neutrophils. The clinical regimens include filgrastim, lenograstim, nartograstim, filgrastim biosimilars, and long-lasting pegylated preparations of filgrastim.[1] The first report regarding G-CSF-associated aortitis was in 2004,[2] and arteritis has been reported as a side effect of G-CSF treatment. However, there are no clear diagnostic criteria or treatment guidelines. Therefore, we report a case of G-CSF-associated aortitis that required careful exclusion of similar aortitis and multiple imaging modalities to support the diagnosis.

Timeline

One month before onset	The 5th course of chemotherapy [paclitaxel 262 mg (180 mg/m^2)/carboplatin 583 mg (target area under the concentration-time curve 6 mg min/mL)] was administered.
Two weeks before onset	Myelosuppression was detected, and the granulocyte colony stimulating factor (G-CSF) was administered for the first time and continued for 3 days.
Ten days before onset	The 6th course of chemotherapy was administered.
Three days before onset	Myelosuppression was detected, and the G-CSF treatment was started and continued for 4 days.
Day 0 (onset)	Last administration of G-CSF. The patient developed a high fever during the night.
Day 1	The patient visited an outpatient clinic and was prescribed acetaminophen and levofloxacin.
Day 4	The patient was admitted to the gynaecology department for a persistent high fever. Although the thickening around the aorta was suspected via computed tomography (CT), it was uncertain whether the inflammation was localized there, and the patient first received cefmetazole for suspected infection or febrile neutropenia.
Day 9	The antibiotic treatment was changed to piperacillin/tazobactam.
Day 16	The fever and inflammation did not improve completely. Aortitis was re-considered as a differential diagnosis of fever. An magnetic resonance imaging was performed to evaluate the aortitis.
Day 17	The patient was transferred to the cardiology department for treatment of the aortitis. Bone marrow testing was performed.
Day 23	Positron emission tomography revealed active inflammation of the aortic arch and abdominal aorta. Prednisolone (PSL) was started (50 mg/day, 1 mg/kg).
Day 24	The fever improved, and the temperature was maintained at <37.5°C.
Day 38	A CT examination revealed that the thickening around the aorta had improved. C-reactive protein concentration returned to normal.
Day 46	The PSL dose was gradually reduced.
Day 71	The patient was discharged (PSL 25 mg/day).
After 9 months	The PSL dose was gradually reduced to 10 mg/day for 9 months.
After 1 year	No episode of infection, and no recurrence of aortitis and cancer. The patient is almost free from the PSL.

Case presentation

A 52-year-old woman underwent six courses of post-operative chemotherapy (paclitaxel 262 mg/carboplatin 583 mg) for ovarian cancer. Myelosuppression was detected after the 5th and 6th chemotherapy courses, which prompted G-CSF treatment (filgrastim, 75 μg/day). The patient developed a high fever after the last G-CSF administration and was admitted 4 days later in the gynaecology department. Negative bacterial, fungal, and viral test results were observed, and a broad-spectrum antibiotic treatment did not completely improve her condition. Aortitis was suspected based on enhanced computed tomography (CT) findings, and she was referred to our cardiology department.

Her peripheral arterial oxygen saturation was 96% (room air), body temperature was 38.0°C with remittent fever, blood pressure was 92/50 mmHg, and bilateral pulse was 70 beats/min. The head and neck had no bruit or tenderness, and the patient reported no visual deterioration or diplopia. Chest auscultation was clear, and cardiac auscultation revealed normal S1 and S2 with no S3 or murmurs. There were no abdominal abnormalities or notable skin lesions and swelling on the extremities. Her medical history included pulmonary embolism and deep vein thrombosis that had been controlled using anticoagulant medication.

Table 1 shows the patient's laboratory findings on admission, which included an elevated C-reactive protein concentration (CRP). Although the white blood cell count (WBC) slightly elevated on admission, it gradually decreased to 1400/μL (normal range 3300–8600). Decreasing of the platelet counts and normocytic anaemia were also observed. Bone marrow tests showed that the three lineages of haematopoietic cells were

Table 1 Laboratory findings on admission (Day 4)

WBC	8780/μL	(3300–8600)	MMP-3	103.8 ng/mL	(17.3–59.7)
Neut	76.0%	(41.8–75.0)	ESR 1.0hr	>140 mm/h	(3.0–15.0)
Lymph	15.7%	(18.5–48.7)	sIL-2R	584 U/mL	(140–394)
Mono	8.1%	(2.2–7.9)	Ferritin	883 ng/mL	(10.0–120.0)
Eo	0.1%	(0.4–8.7)	STS	Negative	
RBC	2.14×10^4/μL	(3.86–4.92)	TPAb	Negative	
Hb	7.2 g/dL	(11.6–14.8)	TbIFN-γ	Negative	
HCT	22.2%	(35.1–44.4)	β-D glucan	<2.50 pg/mL	(-10.99)
Plt	7.0×10^4/μL	(15.8–34.8)	PCT	0.15 ng/mL	(0.00–0.49)
MCV	103.7 fL	(83.6–98.2)	MPO-ANCA	<1.0 U/mL	(-3.4)
MCH	33.6 pg	(27.5–33.2)	PR3-ANCA	<1.0 U/mL	(-3.4)
MCHC	32.4%	(31.7–35.3)	IgA	240 mg/dL	(93–393)
TP	7.0 g/dL	(6.6–8.1)	IgM	74 mg/dL	(50–269)
Alb	3.5 g/dL	(4.1–5.1)	IgG	1468 mg/dL	(861–1747)
UN	12.6 mg/dL	(8.0–20.0)	IgG4	21 mg/dL	(-134)
Cr	0.80 mg/dL	(0.46–0.79)	C3	164 mg/dL	(73–138)
eGFR	59 mL/min/1.73 m^2		C4	31.8 mg/dL	(11.0–31.0)
LDH	170 U/L	(124–222)	CH50	77.1 U/mL	(30.0–53.0)
Na	140 mEq/L	(138–145)	RF	3 U/mL	(0–14)
K	3.8 mEq/L	(3.6–4.8)	FANA	Negative	
Cl	107 mEq/L	(101–108)	APTT	29.9 sec	(23.0–38.0)
CRP	19.39 mg/dL	(0.00–0.14)	PT-INR	1.15	(0.85–1.15)

Anti SS-A/Ro antibody, anti SS-B/La antibodies, anti Sm antibodies, anti-double strand-DNA antibodies, anti-RNP antibodies were all negative. ESR is the data of Day 24. Normal ranges are shown in brackets.

Alb, albumin; APTT, activated partial thromboplastin time; C3, complement component 3; C4, complement component 4; CH50, 50% haemolytic complement activity; Cl, chlorine; Cr, creatinine; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; Eo, eosinophil; ESR, erythrocyte sedimentation rate; FANA, fluorescent anti-nuclear antibodies; Hb, haemoglobin; HCT, haematocrit; Ig, immunoglobulin; K, potassium; Lymph, lymphocyte; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; MCV, mean corpuscular volume; MMP-3, matrix metalloproteinase-3; Mono, monocyte; MPO-ANCA, myeloperoxidase-anti-neutrophil cytoplasmic antibody; Na, sodium; Neut, neutrophil; PCT, procalcitonin; Plt, platelet; PR3-ANCA, serine proteinase3-anti-neutrophil cytoplasmic antibody; PT-INR, international normalized ratio of prothrombin time; RBC, red blood cell; RF, rheumatoid factor; sIL-2, soluble interleukin-2 receptor; STS, serologic test for syphilis; Tb-INF, tuberculosis interferon-gamma; TP, total protein; TPAb, treponema pallidum antibodies; UN, urea nitrogen; WBC, white blood cell.

retained, and there was no increase in blasts or morphological abnormalities.

Electrocardiography, echocardiography, and chest radiography revealed no abnormalities that could explain the fever. Echography of the abdominal aorta revealed aortic wall thickening and a hypoechoic region around the aorta (*Figure 1*). Furthermore, CT revealed an increase in the soft tissue surrounding the aortic arch and abdominal aorta (*Figure 2A,B*). Gadolinium-enhanced magnetic resonance imaging revealed thickening of the aortic wall and enhancement of the wall and perivascular tissue (*Figure 3A*), and uptake was observed during ^{18}F-fludeoxyglucose positron emission tomography (*Figure 3B*). These imaging findings suggested active inflammation at the lesions.

The patient was transferred to the cardiology department, and G-CSF-associated aortitis was suggested based on her history and clinical course (Naranjo adverse drug reaction probability scale score 7; probable). On Day 23, she was exhausted because of the persistent fever and received prednisolone treatment (PSL, 50 mg/day), which promptly resolved her symptoms. Follow-up testing revealed improvement in her CRP concentration and erythrocyte sedimentation rate (Supplementary material online, *Figure S1*). After 2 weeks, CT revealed an improvement in the thickening around the aorta (*Figure 2C*). The PSL dose was gradually reduced. This treatment was marked by the absence of recurrence of the aortitis, cancer, and infection at her 1-year follow-up.

Discussion

This case involved a patient who presented with a significant fever. Aortitis was suspected via multiple imaging modalities, and the association with G-CSF treatment was suggested based on the patient's history and exclusion of other diseases.

Clinical course in previous cases

A PubMed search revealed 28 reported cases involving arteritis associated with G-CSF treatment (*Table 2*). Most cases involved fever at the onset of disease, although other symptoms (e.g. abdominal tenderness, syncope) appeared before the fever in some cases.[5] In this case, PSL was started because of a high fever and malaise; however, one-half of the reported cases resolved without corticosteroids.[6] In the previous cases that involved corticosteroid treatment, three cases were administered high-dose treatments (e.g. pulse methylprednisolone),[4,7] and six cases involved PSL doses starting at 30–60 mg/day with gradual tapering.[3,5] Early reduction of the

Table 2 Previously reported cases

No	Age	Sex	Year	Nationality	Background disease	G-CSF	Symptoms	Lesions	Glucocorticoid treatment
1[2]	55	F	2004	France	Stem cell donor	Filgrastim	Fever, abdominal and lumbar pain, vomiting	Descending ao, abdominal ao	yes
2	54	M	2009	US	Lung cancer	[a]	Fever, epigastric tenderness	Abdominal ao	no
3[3]	52	M	2016	Israel	Healthy donor	Filgrastim	Weight loss, back pain, constipation	Abdominal ao, iliac artery	yes
4	78	F	2016	Japan	Cyclic neutropenia	Filgrastim	Fever, head ache, jaw claudication, visual abnormality	Temporal arteries	yes
5	59	F	2017	Japan	Lymphoma	Pegfilgrastim	Neck and chest pain, fever	Carotid artery, subclavian artery, ao arch, descending ao	yes
6	61	F	2017	Japan	Ovarian cancer	Lenograstim	Fever	Carotid artery	no
7[4]	67	F	2017	Japan	Lung cancer	Pegfilgrastim	Malaise and fever	Carotid artery, thoracic ao	yes
8	61	F	2018	Japan	Breast cancer	Pegfilgrastim	Neck and chest pain → fever	Carotid artery, thoracic ao	no
9	47	F	2018	Japan	Ovarian cancer	[a]	Fever	ao arch, descending ao	yes
10	71	F	2019	Japan	Endometrial cancer	Pegfilgrastim	Fever	ao arch, descending ao	yes
11	72	F	2019	Japan	Lymphoma	Pegfilgrastim	Fever, chest pain	ao arch	no
12	62	F	2019	Japan	Lymphoma	Pegfilgrastim	Fever, chest pain	Descending ao	yes
13	69	M	2019	Japan	Lymphoma	Pegfilgrastim	Fever	Subclavian artery	unknown
14	77	F	2019	Japan	Ovarian cancer	[a]	Fever	Carotid artery, subclavian artery	no
15[5]	60	F	2019	Sweden	Breast cancer	Filgrastim	Abdominal tenderness → fever	Subclavian artery, ao arch, descending ao, abdominal ao	yes
16[5]	70	F	2019	Sweden	Breast cancer	[a]	Syncope, diarrhoea, dehydration → fever	Thoracic ao, brachiocephalic trunk	yes
17	72	F	2019	Japan	Breast cancer	Pegfilgrastim	Fever	Descending ao	no
18[6]	43	F	2020	Japan	Uterine cancer	Pegfilgrastim	Unknown	Thoracic ao	no
19[6]	47	F	2020	Japan	Uterine cancer	Pegfilgrastim	Unknown	Thoracic ao	no
20[6]	74	F	2020	Japan	Tongue cancer	Pegfilgrastim	Unknown	Thoracic ao	no
21[6]	65	F	2020	Japan	Pancreatic cancer	Pegfilgrastim	Fever, chest pain	ao arch, abdominal ao	no
22[7]	66	F	2020	Japan	Breast cancer	Pegfilgrastim	Fever, malaise, abdominal discomfort	ao arch, abdominal ao	yes
23[8]	52	F	2020	Finland	Breast cancer	Filgrastim	Fever, chest pain	Aorta	yes
24[8]	62	F	2020	Finland	Breast cancer	Filgrastim, Pegfilgrastim	Fever	Aorta	yes
25[8]	70	F	2020	Finland	Breast cancer	Lipegfilgrastim	Fever	Aorta, supra-aortic vessels	no
26[8]	56	F	2020	Finland	Breast cancer	Lipegfilgrastim	Fever, neck pain, jaw pain, malaise	Carotid artery, thoracic ao	yes
27[8]	53	F	2020	Finland	Breast cancer	Pegfilgrastim	Fever, sore throat, ear ache, dyspnoea, and chest pain	Aorta	yes
29[8]	40	F	2020	Finland	Breast cancer	Lipegfilgrastim	Fever, sore throat, chest and neck pain, malaise	Carotid artery	yes

ao, aorta or aortic; F, female; M, male.
The figures in square brackets refer to page numbers.
[a]Name of the G-CSF preparations were unknown, however these were used for several days.

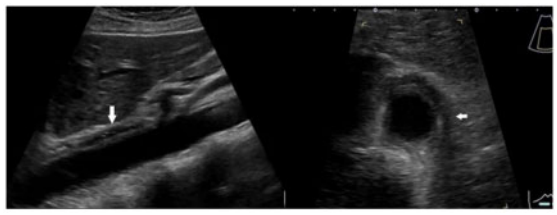

Figure 1 Imaging findings via echography of the abdominal aorta. Echography of the abdominal aorta at the level of the coeliac artery bifurcation (left: long-axis image, right: short-axis image) revealed increased brightness and thickening of the vessel wall. There was a hypoechoic region surrounding the outside of the artery (arrow), and increased blood flow was not observed. Echography helped to distinguish some lesion features (e.g. for an abscess, a tumour, and an atheroma).

corticosteroid may be possible because G-CSF-associated arteritis may have a relatively good prognosis compared to other arteritis.[7] Nevertheless, a case that involved aortic dissection highlighted the need for careful observation.[4] Some cases with G-CSF re-administration had aortitis recurrence,[6] and dose reduction or change of the anti-cancer drug were needed to avoid myelosuppression.

Differential diagnosis of aortitis

Takayasu arteritis and giant cell arteritis (GCA) are immune disorders that cause large-vessel arteritis. Takayasu arteritis onset is most common among women in their 20s, and the lesions are often continuous in the aorta and its primary branches. More than 90% of patients have lesions in the aortic arch, and 40% in the abdominal aorta.[9] Onset of GCA is most common among women in their 60s to 70s, and lesions are typically detected in the branches of the carotid and vertebral arteries, and other large arteries may have lesions.[10] Perivascular inflammation caused by IgG4-related diseases is found in the abdominal and iliac arteries, although lesions can be present in the thoracic aorta. These lesions tend to be detected in men in their sixties.[11] Other differential diagnoses include infection (bacterial, syphilis, human immunodeficiency virus, and tuberculosis), drug-induced disease,[12] malignancy,[13] Behçet disease, Cogan syndrome, systemic lupus erythematosus, and anti-neutrophil cytoplasmic antibody-related vasculitis (Supplementary material online, *Figure S2*).[14] The differential diagnosis needs to be performed based on the characteristics of the diseases in addition to the imaging evaluation.

Lesions and imaging

Previous reports of G-CSF-associated arteritis indicated that most lesions were detected in the thoracic aorta (68%), especially in the arch (29%) and descending aorta (29%). However, lesions can be detected in the abdominal aorta (21%), carotid artery (25%), and subclavian artery (14%) (*Table 2*). Most cases involved circumferential thickening of the peri-arterial tissue, which was detected via CT. Echography was useful in guiding the diagnosis in this case, although none of the previously English reported cases involved an abdominal aorta echography. Nevertheless, an echography was used to evaluate some lesions in the temporal and carotid arteries.[8]

Mechanism of aortitis

The pathological mechanisms underlying G-CSF-associated arteritis are unclear. Previous reports have speculated that arteritis is related to cytokines and complex immune reactions between anti-cancer agents and G-CSF.[8] In aortitis after acute aortic dissection, G-CSF may act on the arterial adventitia and invading granulocytes, which results in inflammation.[15] Nevertheless, this mechanism for G-CSF-associated arteritis remains speculative.

Conclusions

Chronic inflammation can lead to a reduced nutritional status and quality of life in cancer patients, which may influence their general condition and make them unable to continue chemotherapy. When cancer patients experience persistent fever after G-CSF treatment, it is necessary to make a differential diagnosis carefully and select treatments based on the patient's general condition as well as the prognosis of arteries.

Patient perspective

The patient was mentally exhausted before treatment. Psychiatric support also improved her mental status, despite the high dose of PSL used.

Lead author biography

Graduated from Jichi Medical University School of Medicine, Japan and worked as a general physician. In 2016, conducted clinical studies on cardiovascular risk factors among young people at Shinshu University Graduate School of Medicine. Currently working in general cardiology and preventive medicine.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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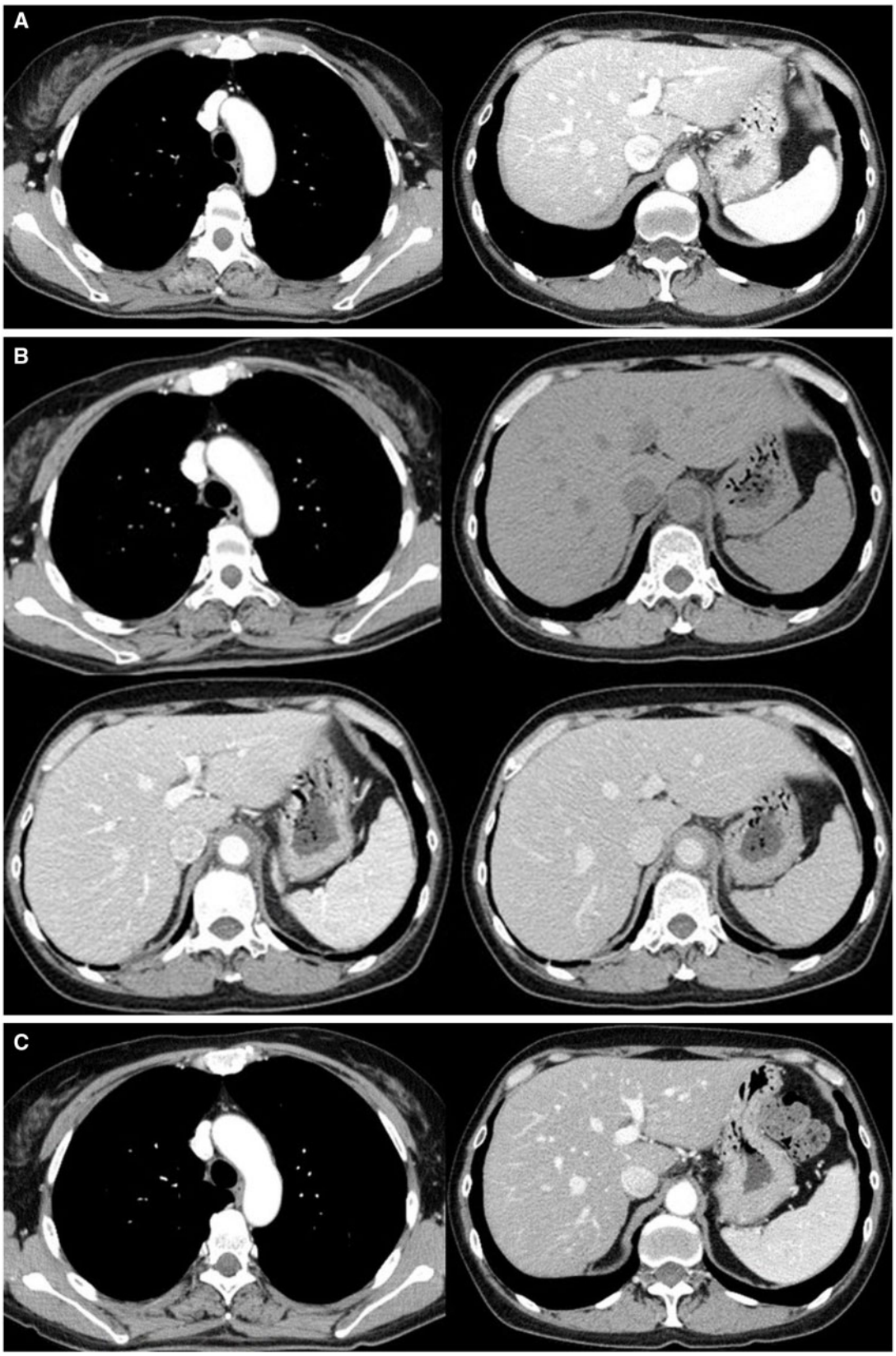

Figure 2 Imaging findings via enhanced computed tomography. (*A*) Enhanced computed tomography (CT) findings before chemotherapy. (*B*) The computed tomography findings after onset (chest: early phase, abdomen: plain, early, and delay phase), which revealed enhancement of the peri-aortic tissue from the aortic arch and the abdominal aorta (vs. the pre-chemotherapy findings). There was no wall thickening in the branches of the aorta, aortic stenosis, aneurism, or dissection. (*C*) Enhanced computed tomography after the treatment for aortitis. The thickening around the aorta had improved after 2 weeks.

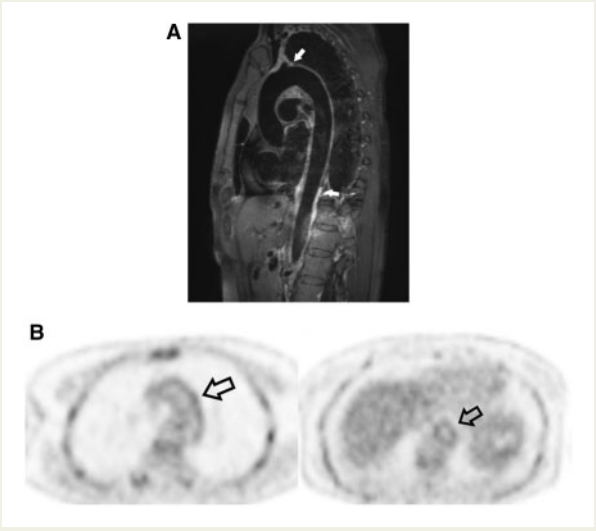

Figure 3 Imaging findings via gadolinium-enhanced magnetic resonance imaging and fluorodeoxyglucose positron emission tomography. (*A*) Gadolinium-enhanced T1-weighted image revealed thickening of the vessel wall at the aortic arch and the abdominal aorta at the diaphragm *(arrow)*. (*B*) Fluorodeoxyglucose positron emission tomography (^{18}F-FDG PET) revealed FDG uptake in the aortic arch and the abdominal aorta, with a standardized uptake value (SUV) of 2–3 like that in the liver (arrow).

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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References

1. Takahashi M, Kondoh C, Takano T. The history of granulocyte-colony stimulating factor (in Japanese). *DDS* 2017;**32**:134–142.
2. Darie C, Boutalba S, Fichter P, Huret JF, Jaillot P, Deplus F *et al.* Aortitis after G-CSF injections (in French). *Rev Med Interne* 2004;**25**:225–229.
3. Miller EB, Grosu R, Landau Z. Isolated abdominal aortitis following administration of granulocyte colony stimulating factor (G-CSF). *Clin Rheumatol* 2016;**35**: 1655–1657.
4. Sato Y, Kaji S, Ueda H, Tomii K. Thoracic aortitis and aortic dissection following pegfilgrastim administration. *Eur J Cardiothorac Surg* 2017;**52**:993–994.
5. Parodis I, Dani L, Notarnicola A, Martenhed G, Fernström P, Matikas A *et al.* G-CSF-induced aortitis: two cases and review of the literature. *Autoimmun Rev* 2019;**18**:615–620.
6. Shirai T, Komatsu H, Sato H, Fujii H, Ishii T, Harigae H. Migratory aortitis associated with granulocyte-colony-stimulating factor. *Intern Med* 2020;**59**: 1559–1563.
7. Mukai T, Kubo S, Morita Y, Yamamoto M, Ikeda M. Aortitis which developed after the administration of granulocyte-colony stimulating factor. *Modern Rheumatol Case Rep* 2020;**4**:74–78.
8. Taimen K, Heino S, Kohonen I, Relas H, Huovinen R, Hänninen A *et al.* Granulocyte colony-stimulating factor- and chemotherapy-induced large-vessel vasculitis: six patient cases and a systematic literature review. *Rheumatol Adv Pract* 2020;**4**:1–10.
9. Isobe M. Diagnosis of large-vessel and medium-vessel vasculitis: a new guideline for management of vasculitis syndrome (in Japanese). *Jpn J Imaging Diagn* 2019;**39**: 146–153.
10. Lie JT. Aortic and extracranial large vessel giant cell arteritis: A review of 72 cases with histopathologic documentation. *Semin Arthritis Rheum* 1995;**24**: 422–431.
11. Mizushima I, Kasashima S, Fujinaga Y, Notohara K, Saeki T, Zen Y *et al.* Clinical and pathological characteristics of IgG4-related periaortitis/periarteritis and retroperitoneal fibrosis diagnosed based on experts' diagnosis. *Ann Vasc Dis* 2019;**12**:460–472.
12. Mettler C, Chouchana L, Terrier B. Antineoplastic drug-induced aortitis: an unraveled adverse effect using the World Health Organization pharmacovigilance database. *J Rheumatol* 2020;**47**:1298–1300.
13. Lopez FF, Vaidyan PB, Mega AE, Schiffman FJ. Aortitis as a manifestation of myelodysplastic syndrome. *Postgrad Med J* 2001;**77**:116–118.
14. Hartlage GR, Palios J, Barron BJ, Stillman AE, Bossone E, Clements SD *et al.* Multimodality imaging of aortitis. *JACC Cardiovasc Imaging* 2014;**7**:605–619.
15. Anzai A, Shimoda M, Endo J, Kohno T, Katsumata Y, Matsuhashi T *et al.* Adventitial CXCL1/G-CSF expression in response to acute aortic dissection triggers local neutrophil recruitment and activation leading to aortic rupture. *Circ Res* 2015;**116**:612–623.