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Case Report

Role of Glucocorticoids in Resolution of Granulocyte Colony Stimulating Factor–Induced Aortitis: 2 Cases and Review of the Literature

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Abstract

Recombinant human granulocyte colony-stimulating factor (G-CSF) is widely established as primary or secondary prophylaxis against neutropenia for patients undergoing chemotherapy. Despite the common side effects being generally mild, some rare but severe adverse events have been documented. In recent years, increasing numbers of G-CSF-induced aortitis have been reported worldwide. However, clinicians have not yet reached a consensus on the management and treatment of this condition.

In this report, previously reported cases of G-CSF-induced aortitis in the literature are reviewed, and 2 additional independent cases are presented that share multiple common features regarding patient gender, age, G-CSF treatment regimen, imaging modalities for diagnosis, and disease course. In one of the cases, the patient was treated empirically with glucocorticoids, while in the other case, no specific treatment was given for the aortitis other than discontinuation of the G-CSF treatment. In both cases, improvement of symptoms and complete resolution of the inflammation were achieved about 1 month after the disease onset.

Aortitis is a rare but severe adverse event of G-CSF therapy and should be suspected upon relevant complaints, prompting rapid investigation. Although empirical use of glucocorticoids appears efficient for the resolution of the inflammation and symptoms, no substantial differences in the disease course were obtained in comparison to spontaneous remission upon discontinuation of G-CSF.

Keywords: Adverse events, aortitis, cancer, case report, G-CSF, glucocorticoids

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Introduction

Inflammation of the vessel walls of arteries is referred to as aortitis. Hence, aortitis is a type of large vessel vasculitis, commonly occurring on an autoimmune basis and affecting the thoracic aorta, but also other large vessels such as the abdominal aorta and proximal limb arteries may be affected.[1,2] The disease aetiology remains largely unknown; however, 1 of the 2 main subgroups, giant cell arteritis (GCA), is more common in populations of Scandinavian origin, among adults aged 50 or older, and the female-to-male ratio is generally estimated to be 3 : 1.[3,4]

Recombinant human granulocyte colony-stimulating factors (G-CSF; filgrastim and pegfilgrastim) reduce the incidence and severity of neutropenia in patients at risk of infections and neutropenic fever.[5,6] The G-CSF treatment is widely evaluated and established as a primary or secondary prophylaxis for patients undergoing chemotherapy, and an adequate safety profile has been documented.[5-8] In addition, reduction of all-cause mortality has been shown in a randomized controlled trial with dose-dense G-CSF support for chemotherapy.[9] Common side effects of G-CSF treatment include fever, fatigue, musculoskeletal pain, anemia, and nausea. In addition, some rare but serious conditions such as fatal lung injuries, capillary leak syndrome, and arterial thrombosis have also been reported.[10-12] Lastly, hematological malignancy,[13] splenomegaly, and splenic rupture[14] related to G-CSF treatment have been discussed. Based on the knowledge, 2 cases of G-CSF-induced aortitis from Sweden have previously been reported,[15] and in recent years, the number of reported cases has increased worldwide,[16] urging more attention to this condition. Two additional independent cases of G-CSF-induced aortitis in patients who received G-CSF as an adjunct to chemotherapy are reported, and the role of glucocorticoids in the resolution of this condition is discussed based on clinical experience and reports in the literature.

Case Presentation

Case 1

A 74-year-old female presented to the emergency department 18 days after having received her third dose of chemotherapy with carboplatin and paclitaxel due to syncope, cramps, fever, and diarrhea. She had been medicated with filgrastim (Zarzio®) injections on day 10, day 12, and day 14 after the chemotherapy. Her medical history included an appendectomy at the age of 10, osteoporosis, osteoarthritis in the left knee, branch duct intraductal papillary mucinous neoplasms, and a right-sided breast cancer diagnosed and operated in 2002, followed by postoperative chemotherapy, radiotherapy and hormonal therapy with tamoxifen. In 2004, she suffered from a local relapse of her breast cancer and was successfully treated with mastectomy and postoperative cytostatic therapy. Despite normal inner genitalia upon examination, she was diagnosed in 2020 with a peritoneal adenocarcinoma originating from the ovary. She had also been tested negative for breast cancer susceptibility gene (BRCA) 1 and 2 mutations.

The physical examination revealed fever and dehydration, without any other abnormalities. Laboratory tests showed low hemoglobin, hyponatremia, hyperkalemia, and elevated C-reactive protein (CRP) levels (Figure 1A). The neutrophil count was elevated (13 100 cells/μL [normal range: 1600-5900 cells/μL]). The patient was admitted to the hospital for observation; however, she developed fever up to 40°C later during the same day and was initiated on intravenous antibiotics (4 g/0.5 g of piperacillin with tazobactam 3 times a day).

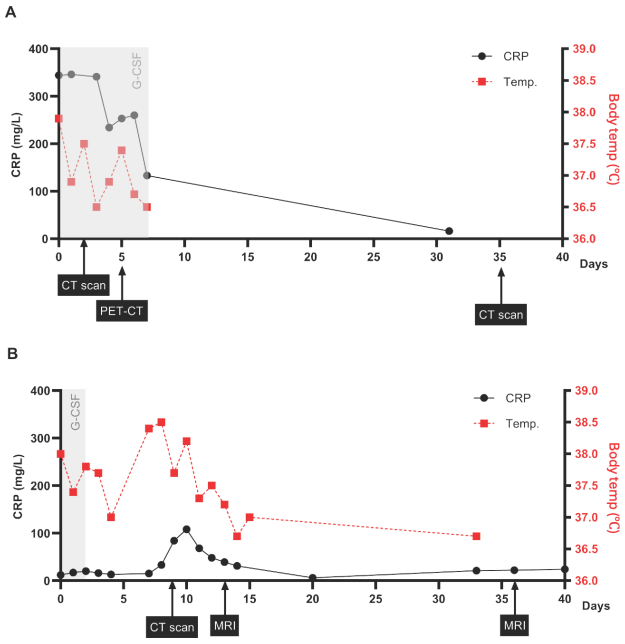

Figure 1. CRP and body temperature over time. A: Timeline of serum C-reactive protein (CRP) levels and body temperature for the patient case 1. B: Timeline of serum CRP levels and body temperature for the patient case 2. P1: Patient 1; P2: Patient 2; G-CSF: recombinant human granulocyte colony-stimulating factor. Day 0 indicates the day of the first admission to the hospital. The shadowed areas indicate time on G-CSF therapy.

Main Points

- Two independent cases of granulocyte colony-stimulating factor (G-CSF)-induced aortitis from Sweden in patients undergoing chemotherapy are reported, and the observed findings are discussed in relation to current literature.
- The cases hold several common features, including the patients' female sex, as well as similar age, medical history, symptoms, and degree of inflammation.
- Upon confirmation of aortitis, direct discontinuation of the G-CSF therapy is essential. By contrast, while glucocorticoids appear to be effective toward rapid resolution of the inflammation and symptoms of aortitis, it might not be a necessary treatment in all cases.

The patient also complained about new-onset pain located between her shoulders and at the epigastrium. A computed tomography (CT) scan was conducted where the pulmonary artery and aorta were visualized, followed by a positron emission tomography-CT (PET-CT) scan; both revealed thickening of the tunica of the descending aorta, arcus aorta and carotid artery, changes indicative of large vessel vasculitis that were not present in previous imaging. The antibiotics were discontinued on day 4 from commencement due to negative cultures. Following results from the radiological examinations, filgrastim, which had been administered for a total of 3 doses prior to the hospitalization, was suspected as a cause for the large vessel vasculitis and was discontinued. Instead, the patient was initiated on oral prednisolone 50 mg/day for 3 weeks with a subsequent tapering schedule. Two days after commencement of the glucocorticoid therapy, the patient showed rapid clinical improvement, and her CRP as well as body temperature started to slowly decrease (Figure 1A). The patient was discharged after 8 days of inpatient care. She continued with her chemotherapy with carboplatin and paclitaxel as planned, but without the addition of filgrastim.

A follow-up CT of the thorax and abdomen was performed 34 days after admission, revealing resolution of the aortitis. At the same time, the serum CRP level was 16 mg/L (normal range <3 mg/L). Two months later, the serum CRP levels were normalized (1.6 mg/L). The total duration of glucocorticoid therapy for the aortitis was 8.5 months. No relapse of the aortitis had been documented until the last available documentation, which corresponds to 34 months from the aortitis onset.

Case 2

A 62-year-old female sought emergency medical care due to fever after having been medicating with filgrastim (Accofil®). Her medical history included an appendectomy in childhood, and a left-sided invasive ductal mammary cancer stage II with estrogen receptor 95%, progesterone receptor 40%, marker of proliferation Ki-67 21%, and human epidermal growth factor receptor 2 positive with immunohistochemistry 2+, diagnosed 4.5 months prior to symptom onset. She was treated with partial mastectomy with sentinel node assessment, followed by postoperative cytostatic therapy with epirubicin and cyclophosphamide (E90C), docetaxel and trastuzumab, as well as filgrastim as an adjunct for prevention of neutropenia.

The last chemotherapy cycle was given 5 days before the onset of symptoms that led to the diagnosis of aortitis. The initial clinical examination at the emergency room revealed fever 38°C and a new-onset heart murmur (grade 3 of 6) with punctum maximum in the

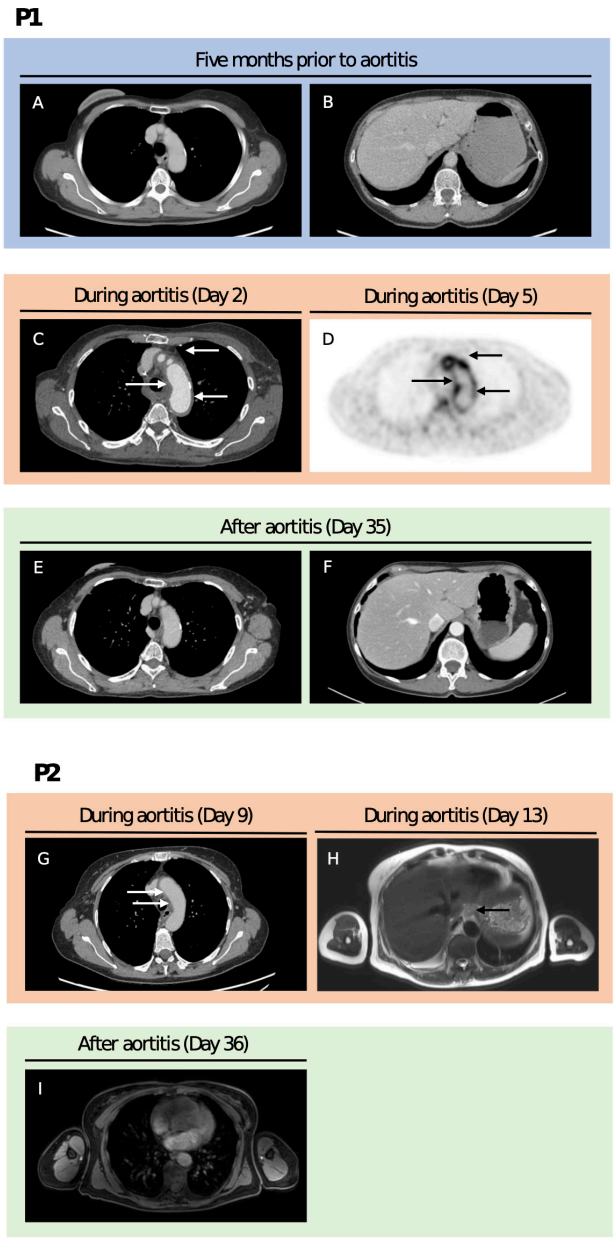

Figure 2. Imaging over time. This figure delineates imaging examinations over time for patient case 1 (A-F) and patient case 2 (G-I). A: Thoracic CT scan without aortic wall thickening prior to the aortitis event. B: Abdominal CT scan without aortic wall thickening prior to the aortitis event. C: Thoracic CT scan showing thickening of tunica of the aortic wall at the level of the aortic arch. D: PET-CT with FDG uptake in the aortic wall at the level of the aortic arch. E: Follow-up thoracic CT scan showing resolution of the aortic wall thickening at the level of the aortic arch. F: Follow-up abdominal CT scan without aortic wall thickening. G: Thoracic CT scan showing signs of vasculitis in the aorta. H: MRI with inflammatory changes in the perivascular region. I: Follow-up MRI showing resolution of the inflammatory findings. Arrows point to pathological findings. P1: Patient 1; P2: Patient 2.

second intercostal space to the right of the sternum. Laboratory tests showed a slightly elevated serum CRP level (12 mg/L, normal range <3 mg/L), white blood cell count 500 cells/μL, of which the neutrophil count was <100 cells/μL. The patient fulfilled the criteria for a neutropenic fever and was therefore admitted to an inpatient care unit. Initially, she received 4 g piperacillin with 0.5 g tazobactam intravenously 4 times a day, and filgrastim was discontinued from day 3 in the ward, with respect to the potential side effects of G-CSF. The patient was clinically stable, the blood cultures were negative, the white blood cell count showed an increasing trend, and her heart murmurs were attributed to dehydration and anemia. Hence, the patient was discharged after 5 days of inpatient care with prescriptions of oral antibiotics (amoxicillin and clavulanic acid) for a total of 10 days of treatment.

Only 3 days later, the patient was re-admitted to the hospital due to fever and chills despite oral antibiotics. In addition, she complained of new-onset back pain. Physical examination showed persistent heart murmurs and facial rashes. In laboratory tests, serum CRP had increased from 15 to 33 mg/L, hemoglobin was still low, and no signs of neutropenia were documented. Despite resuming the medication with piperacillin/tazobactam, the patient had persistent fever. An echocardiography and CT scan of the thorax disclosed thickening of the anterior mitral valve and signs of vasculitis in the aorta. Four days later, magnetic resonance imaging (MRI) of the thoracic arteries confirmed pronounced inflammatory changes in the perivascular region in the brachiocephalic trunk and proximal right common carotid artery, as well as multiple suspected stenoses along the right axillary artery and right brachial artery. Further examinations revealed no indications of other inflammatory or rheumatic diseases. The patient was clinically stable, with gradually decreasing inflammatory parameters (Figure 1B) and was hence discharged after having stayed in the ward for 8 days.

Within the following month, the patient sought emergency care 2 additional times due to fever, dry cough, and pain between the shoulders. The laboratory test results were unchanged on both occasions; serum CRP was 21 and 24 mg/L at the first and second visit, respectively. A follow-up MRI 1 month after the initial admission indicated a clear resolution of the vasculitis findings. It is worth noting that no glucocorticoid treatment had been administered. This was due to the spontaneous resolution of symptoms and radiological findings within a relatively narrow window of time during the investigational process. After 2 additional months, no signs of vasculitis or stenoses could be detected in a follow-up MRI. No relapse of the aortitis had been documented until the last available documentation, which corresponds to 45 months from the aortitis episode.

Discussion

Large vessel vasculitis is generally seen in patients aged 50 years or older and is characterized by a predominance of female sex.[1,17] The clinical presentation of GCA is highly diverse, oftentimes encompassing general symptoms of systemic inflammation such as fever, weight loss, anemia, fatigue, joint pain, and elevated inflammatory parameters.[1,3] Organ-specific

manifestations are likely related to ischemic injury, including skin rashes, headaches, visual impairment, and jaw claudication.[3] The G-CSF mediates maturation and differentiation of neutrophils, stimulates inflammatory cytokine production, and is commonly used to facilitate maintenance of the intensity of chemotherapy while preventing complications, especially neutropenic fever.[6,8,9] Side-effects of G-CSF treatment are generally mild; however, attention on and knowledge about G-CSF-induced aortitis, a rare but serious adverse event, is much needed.

In this report, 2 female patients aged 74 and 62 years who developed aortitis verifiable on radiographic imaging after receiving G-CSF, administered in parallel with chemotherapy are described. Both patients sought emergency care due to fever, presented systemic symptoms and elevated inflammatory markers, and complained about new-onset musculoskeletal pain in the back during their time in the ward. With respect to the medical history, both patients reported here had a primary cancer disease (Patient 1 (P1): peritoneal adenocarcinoma originating from the ovary; Patient 2 (P2): breast cancer) and were undergoing postoperative chemotherapy. Filgrastim was administered in both cases prior to their visit to the emergency department. Both patients had a history of appendectomy during childhood. Moreover, P1 was previously successfully treated for breast cancer despite 1e relapse, while P2 had no previous history of malignancy prior to the current episode.

Table 1. Comparison Across Reported Cases of G-CSF-associated Aortitis from the Karolinska University Hospital in Stockholm, Sweden

Characteristic	P1	P2	Ref (15) P1	Ref (15) P2
Gender	Female	Female	Female	Female
Age	74	62	70	60
Primary cancer disease	Peritoneal AC of ovarian origin	Breast cancer	Breast cancer	Breast cancer
Chemotherapy agent(s)	Paclitaxel, carboplatin	Docetaxel, trastuzumab, E90C	Docetaxel, trastuzumab, pertuzumab	Docetaxel
G-CSF	Filgrastim	Filgrastim	Filgrastim	Filgrastim
Onset from G-CSF initiation	22 days	3 months	9 days	11 days
Symptoms at emergency department	Syncope, diarrhoea, cramps, fever	Fever	Syncope, diarrhoea, dehydration	Abdominal pain
Aortitis, year of diagnosis	2021	2019	2018	2018
Aortitis, location	Thoracic	Right brachiocephalic trunk, common carotid artery, axillary, and brachial	Thoracic	Thoracic
Imaging modality (for diagnosis)	CT, PET-CT	CT, ECHO	CT	CT
Imaging modality (follow-up)	CT	MRI	PET-CT	PET-CT
Glucocorticoid treatment	Yes	No	Yes	Yes
Resolution of symptoms	Rapid, complete	Rapid, complete	Rapid, complete	Rapid, complete

AC, adenocarcinoma; CT, computed tomography; ECHO, echocardiography; G-CSF, granulocyte colony-stimulating factor; MRI, magnetic resonance imaging; PET, positron emission tomography; P1, patient 1; P2, patient 2.

A comparison of the patients presented herein with the 2 cases from Sweden previously reported by Parodis et al[15] reveals multiple common characteristics regarding the medical history and disease course (Table 1). All 4 patients are females aged 60-74 years, received variants of taxanes (paclitaxel, docetaxel) as a monotherapy or in combination with other agents, were supported with G-CSF, and suffered from aortitis in the thoracic region. These similarities may indicate an elevated risk for developing G-CSF-induced aortitis in elder females treated with taxane; however, due to few reported cases in Sweden, which limits us from detailed data analysis, this distribution could also be explained by G-CSF being more commonly prescribed to elder patients undergoing chemotherapy, or by the fact that females are at a higher risk of GCA than males.[3] On the other hand, in a multinational perspective, 91.8% of the reported cases (n = 45) were female, and 79.6% were patients aged 50 years or older,[16] corroborating a pattern comparable to what is described herein. Regarding the type of G-CSF, filgrastim was used in all cases from Sweden, while pegfilgrastim, the PEGylated, long-acting type of G-CSF, may be more commonly used in other countries.[15,16] Based on data in the current literature, it is yet unclear whether 1 of these 2 types of G-CSF is more likely to induce aortitis.[16,18-21]

Setting the clinical presentations side by side as illustrated in Table 1, all patients reported symptoms of systemic inflammation, and the ones not presenting with fever at the emergency room developed fever shortly after admission. However, the time to the onset of aortitis appears to vary. While patients in 3 of the 4 cases from Sweden suffered from aortitis within 22 days from G-CSF initiation, the inflammation onset occurred 3 months after G-CSF commencement in one of the cases. This observation has clinical implications, as it may help increase awareness and direct the physician's suspicions toward G-CSF-induced aortitis when patients who recently commenced a treatment regimen that encompasses G-CSF seek care due to general inflammatory symptoms, thereby reducing delay. At the same time, it is worth noting that the onset of inflammation does not seem to necessarily occur shortly after the initiation of G-CSF therapy. Also, the relatively late onset in the cases, especially in one of those, might be due to a subclinical evolution phase before the development of overt symptoms, making G-CSF-induced aortitis difficult to diagnose and the chronological associations less apparent.

Aortitis is diagnosed through a combination of clinical assessment, imaging, and biopsy findings.[3,16,22] Apart from clinical assessment, CT scan was employed as a diagnostic modality in all 4 cases, even though PET-CT or MRI were also performed to confirm the findings for the

patients in the present report. While CT is often used for initial imaging assessment due to its high availability and spatial resolution, which makes the modality suitable for the assessment of the thickness and regularity of the aortic wall as well as inflammation in the soft tissues,[23] MRI visualizes discrete signs of early inflammation in the vessels as well as changes during later phases, also constituting a better choice for younger patients or for follow-ups since it does not involve ionising radiation.[23,24] The PET-CT characterizes tissue with higher metabolism and is useful in the assessment of inflammatory activity or when malignancy is suspected, although it has a limited spatial resolution compared to the other 2 modalities.[25] While the use of multiple expensive imaging techniques may be questioned from the perspective of cost-effectiveness, combination of PET-CT with conventional CT scans has been suggested to increase the sensitivity and specificity of the examination of the aortic wall.[23,25]

Regarding treatment of the aortitis, all 4 patients were at first treated with broad-spectrum antibiotics (piperacillin/tazobactam), and glucocorticoid treatment was initiated in 3 of the 4 cases from Sweden upon identification of signs of aortitis. Patients who received glucocorticoids responded rapidly and completely, while P2 in the present report fully recovered upon discontinuation of the G-CSF therapy, without glucocorticoid treatment. As also mentioned in the report by Parodis et al,[15] 1 patient had shown partial regression regarding the thickness of the aortic wall before commencing glucocorticoids. Since treatment with glucocorticoids is commonly used for GCA,[23] its empiric use in G-CSF-induced aortitis has been considered a reasonable strategy; however, a case of spontaneous regression and remission following discontinuation of the contributing agent is presented. Thus, tentative surveillance may be considered in some non-severe cases rather than direct commencement of glucocorticoid therapy, in light of the well-acknowledged long-term adverse effects of glucocorticoids.[26-28] Neither in the patients reported here nor in previous publications[15,16] were substantial differences seen between patients who received glucocorticoids and the patient who did not with regard to disease course or duration of the inflammation or symptoms, from disease onset to remission. Overall, further investigation is needed to determine the value of glucocorticoids in G-CSF-induced aortitis.

Summary and Concluding Remarks

In the present report, 2 independent cases of G-CSF-induced aortitis from Sweden in patients undergoing chemotherapy are described, and the observed findings are discussed in relation to the current literature. The cases hold several common features, including the patient's gender, age, medical history, symptoms, and degree of inflammation. Increased awareness of aortitis as a rare but severe complication of G-CSF therapy should be acquired and effectuated, and relevant complaints should prompt rapid investigation. Upon confirmation of aortitis, direct discontinuation of the G-CSF therapy is essential. In addition, while glucocorticoids appear to be effective in the process toward rapid resolution of the inflammation and symptoms of aortitis, it might not be a necessary treatment in all cases.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – N.P., A.B., L.D., O.P.B.W., I.P.; Design – N.P., A.B., L.D., O.P.B.W., I.P.; ; Supervision – N.P., A.B., L.D., O.P.B.W., I.P.;; Resources – N.P., A.B., L.D., O.P.B.W., I.P.; Materials – N.P., A.B., L.D., O.P.B.W., I.P.; Data Collection and/or Processing – N.P., A.B., L.D., O.P.B.W., I.P.; Analysis and/or Interpretation – N.P., A.B., L.D., O.P.B.W., I.P.; Literature Search – N.P., A.B., L.D., O.P.B.W., I.P.; Writing Manuscript – N.P., A.B., L.D., O.P.B.W., I.P.; Critical Review – N.P., A.B., L.D., O.P.B.W., I.P.

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