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Rapidly Progressive Ascending Aortic Aneurysm Associated With Suspected Giant Cell Arteritis: An Urgent Cardiac Surgical Case

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Skander HAFI [1], Jan Remes [1], Arnaud Henkens [1], Silvia Solari [1]

1. Cardiac Surgery, Centre Hospitalier Universitaire de Saint-Pierre, Brussels, BEL

Corresponding author: Skander HAFI, skander.hafi@ulb.be

Abstract

Giant cell arteritis (GCA) may involve the thoracic aorta and cause life-threatening vascular complications. We report an 80-year-old man with polymyalgia rheumatica (PMR) who developed palpitations, dizziness, and intermittent dyspnea. Contrast-enhanced computed tomography (CECT) angiography showed an ascending aortic aneurysm measuring up to 78 mm, compared with an approximately 40 mm diameter on a non-contrast chest CT one year earlier, along with focal aortic wall ulceration. Urgent ascending aortic replacement was undertaken using a 32 mm Dacron graft. Inflammatory changes rendered dissection of the aneurysmal segment technically challenging. Histopathology demonstrated lymphocytic aortitis involving the intima, media, and adventitia, raising suspicion of GCA-associated aortic disease. Postoperative hemorrhage required re-exploration on day one, and atrial fibrillation was treated medically. The patient was discharged on postoperative day seven. Fluorodeoxyglucose-positron emission tomography/computed tomography (FDG-PET/CT) at 47 days showed no metabolic evidence of active large-vessel vasculitis. Rheumatologic review at approximately eight months documented clinical remission of PMR under methotrexate and hydrocortisone. This case highlights urgent surgical management of a rapidly enlarging ascending aortic aneurysm and the diagnostic value of histopathological examination of resected aortic tissue.

Categories: Cardiology, Rheumatology, Cardiac/Thoracic/Vascular Surgery
Keywords: aortitis, ascending aortic aneurysm, cardiac surgery, giant cell arteritis, polymyalgia rheumatica

Introduction

Giant cell arteritis (GCA) is the most common large-vessel vasculitis in adults older than 50 years and is increasingly recognized as a systemic disease extending beyond its classic cranial manifestations [1]. Involvement of the thoracic aorta and its branches may remain clinically silent until aneurysm formation, dissection, or rupture occurs [2,3]. Chronic vascular inflammation may cause structural damage to the aortic wall and predispose to aneurysmal degeneration [4].

Polymyalgia rheumatica (PMR) and GCA are closely related inflammatory conditions. Large-vessel involvement is increasingly identified in GCA and less frequently in patients with PMR; clinically significant vascular complications are particularly associated with GCA [5]. Suspected GCA is evaluated using clinical findings, temporal artery biopsy or ultrasound, and vascular imaging including computed tomography angiography, magnetic resonance angiography, or fluorodeoxyglucose-positron emission tomography (FDG-PET), as appropriate [6,7]. The 2022 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) criteria are classification rather than diagnostic criteria [8].

In contrast to the present case, nonsyndromic ascending aortic aneurysms usually enlarge slowly: growth rate of 0.2 ± 0.4 mm/year. From a cardiac surgical perspective, inflammatory aortitis may increase operative complexity, while the diagnosis itself is sometimes made only after histopathological examination of the resected aorta [4]. This report describes an exceptionally large interval change in ascending aortic diameter, urgent supracoronary replacement, and the subsequent identification of lymphocytic aortitis in a patient with PMR.

Case Presentation

Clinical presentation and initial assessment

An 80-year-old man with PMR diagnosed in mid-2023 presented with intermittent palpitations and dizziness evolving over approximately one week. At an emergency department visit during the end of 2025, he denied chest pain, dyspnea, headache, and fever. During cardiologic reassessment, he reported episodic palpitations with presyncope and breathlessness but no chest or back pain. His history included arterial hypertension, dyslipidemia, and type 2 diabetes. PMR had previously been investigated by FDG-PET in mid-2023, showing inflammatory uptake in the shoulder and pelvic girdles without reported large-vessel vasculitis. He had received methotrexate and glucocorticoids before the aortic presentation.

The available documentation did not include separate systolic pressures in both arms, a systematic examination of upper-limb pulses or bruits, temporal artery findings, or a complete cranial GCA symptom inventory. These findings are therefore not reported as negative. The preoperative ECG showed sinus rhythm at approximately 65 beats/min, first-degree atrioventricular block (PR 252 ms), and right bundle branch block (QRS 142 ms). The preoperative C-reactive protein concentration was 1.2 mg/L during the end of 2025; a contemporaneous erythrocyte sedimentation rate was not identified in the available record.

Transthoracic echocardiography showed preserved left ventricular systolic function, without a quantified ejection fraction, and marked ascending aortic dilatation beginning beyond the sinotubular junction. Very mild aortic regurgitation was present, without major valve dysfunction. Aortic valve morphology was subsequently described as tricuspid on CT. Preoperative coronary angiography on end of 2025 showed only non-obstructive coronary disease, including a proximal left anterior descending artery plaque of less than 30%. Carotid and vertebral duplex ultrasonography showed no hemodynamically significant stenosis.

Aortic imaging and surgical indication

A non-contrast, non-ECG-gated chest CT obtained during the end of 2024, during pulmonary evaluation showed an ascending aortic diameter of approximately 40 mm on retrospective assessment (Figure *1A*).

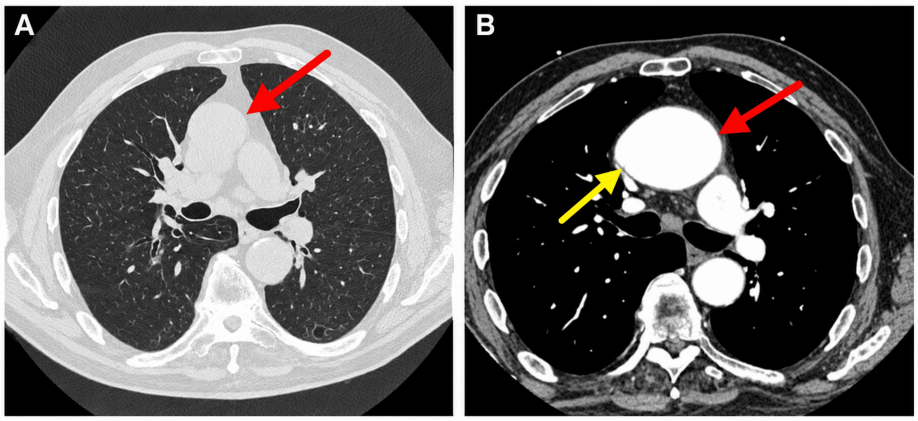


FIGURE 1: Interval enlargement of the ascending aorta between the end of 2024 and a year later

(A) Non-contrast, non-ECG-gated chest CT performed during end 2024, showing an ascending aortic diameter retrospectively estimated at approximately 40 mm (red arrow); (B) ECG-gated contrast-enhanced CT angiography performed a year later, demonstrating marked aneurysmal dilatation of the ascending aorta, measuring approximately 78 mm (red arrow), with a focal aortic wall abnormality (yellow arrow). The two examinations were performed using different acquisition protocols, limiting direct quantitative comparison of aortic diameters.

The contemporaneous radiology report did not explicitly provide this aortic measurement. A year later, ECG-gated contrast-enhanced computed tomography (CECT) angiography demonstrated marked enlargement of the ascending aorta to 60 × 76 mm (Figure *1B*); the surgical documentation described the maximum diameter as approximately 78 mm. The sinuses of Valsalva measured 27 × 38 mm, the sinotubular junction 43 × 46 mm, the aortic arch 30 × 42 mm, and the descending thoracic aorta 31 × 33 mm. The CT report described a focal recess adjacent to the superior vena cava, while the surgical team documented a focal ulcerated lesion of the aortic wall.

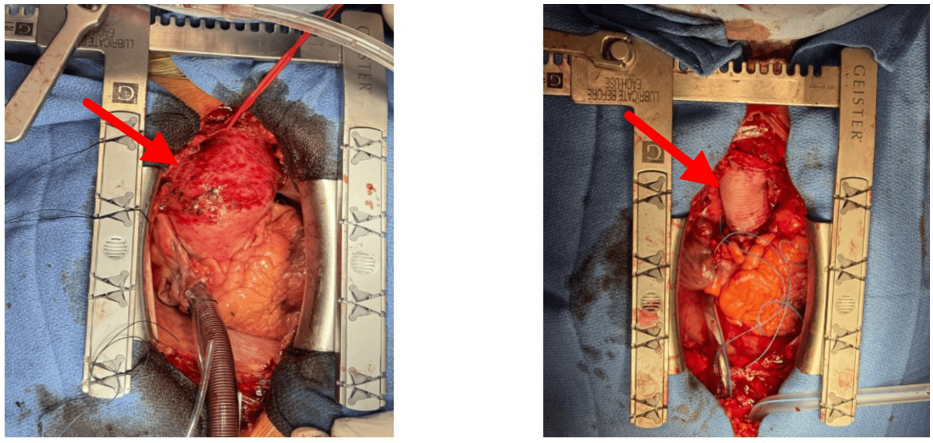
The apparent interval increase was substantial compared with previously reported growth rates for nonsyndromic ascending aortic aneurysms [9]. However, precise annual growth cannot be calculated confidently because the baseline and follow-up CT examinations used different acquisition protocols and standardized centerline measurements at an identical anatomical level were not documented [10]. The available imaging reports did not definitively distinguish a penetrating atherosclerotic ulcer from inflammatory ulceration or focal pseudoaneurysmal change, nor did they characterize the aneurysm as unequivocally fusiform or saccular. There was no documented acute aortic dissection. The patient's marked aneurysm size, focal ulceration, and rapid apparent enlargement prompted admission for intensive blood-pressure control and expedited preoperative evaluation. The operation was performed urgently during the end of 2025, two days after the diagnostic CECT study, rather than as an immediate resuscitative emergency [11].

As part of the preoperative assessment, an extensive etiological workup was performed to investigate potential infectious causes of aortic disease. Infectious investigations, including bacterial, syphilitic, mycobacterial, viral, and fungal screening, were negative, making an infectious etiology unlikely. Given the

patient's history of PMR and the absence of evidence supporting an infectious origin, an underlying noninfectious inflammatory aortic process was considered.

Surgical management

Urgent ascending aortic replacement was performed through median sternotomy. After opening the pericardium, a moderate serosanguineous effusion was observed. Femoral arterial and right atrial venous cannulation were used to establish normothermic cardiopulmonary bypass. Intraoperative inspection demonstrated pronounced aneurysmal enlargement and an abnormal aortic wall with focal ulceration (Figure *2A*).



(A) (B)

FIGURE 2: Intraoperative findings during ascending aortic replacement

(A) Intraoperative view showing the markedly dilated ascending aortic aneurysm before resection (red arrows); (B) Completed supracoronary replacement of the ascending aorta using a 32-mm Dacron graft (red arrow).

Dissection of the aneurysmal ascending aorta was technically challenging because of the surrounding inflammatory changes, which complicated separation of the tissue planes. Meticulous dissection was therefore required to mobilize the diseased aorta toward the aortic arch.

After aortic cross-clamping and administration of cold blood cardioplegia, the aneurysmal ascending aorta was resected and replaced with a 32 mm Dacron graft. Proximal and distal anastomoses were constructed using 4-0 polypropylene sutures reinforced with felt strips, with surgical sealant applied to the suture lines. The completed ascending aortic replacement is shown in Figure *2B*. The supracoronary distribution of disease and absence of significant aortic valve dysfunction supported preservation of the native aortic root and valve. The resection extended distally toward the aortic arch. Cardiopulmonary bypass lasted 162 minutes and aortic cross-clamping 123 minutes. There was no circulatory arrest. Separation from bypass was uncomplicated, and no complication was reported during the initial operation (Table *1*).

Parameter	Finding
Date / priority	End of 2025 / urgent
Approach	Median sternotomy
Arterial / venous cannulation	Femoral artery / right atrium
CPB temperature	Normothermia
Myocardial protection	Cold blood cardioplegia
CPB duration	162 minutes
Aortic cross-clamp duration	123 minutes
Circulatory arrest	No
Repair	Supracoronary ascending aortic replacement
Graft	32 mm Dacron
Anastomotic reinforcement	Felt strips and sealant
Intraoperative finding	Moderate serosanguineous pericardial effusion
Initial intraoperative complications	No
Postoperative re-exploration	Postoperative day one; hemorrhage

TABLE 1: Operative characteristics

CPB: cardiopulmonary bypass.

Histopathology

The resected aortic specimen was submitted for histopathological examination (Figure 3).

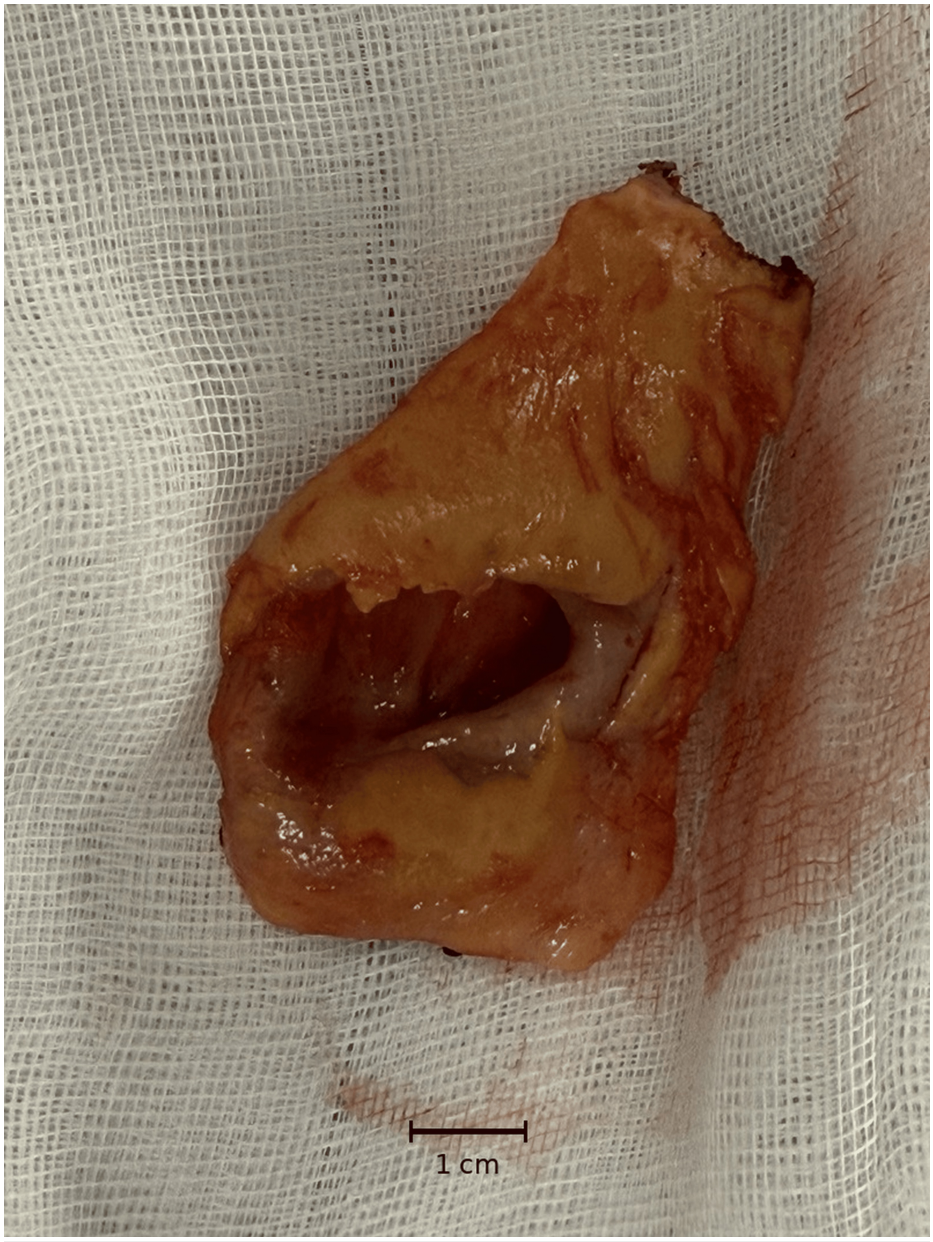


FIGURE 3: Resected ascending aortic specimen

Gross surgical specimen with focal ulcerated area as identified on the original photograph.

The formal pathology report described an unoriented specimen measuring 7.5 × 4 × 0.3 cm. Microscopy demonstrated a mixed inflammatory infiltrate predominantly composed of lymphocytes involving the adventitia, media, and intima. Cluster of differentiation 3 (CD3) immunostaining confirmed T lymphocytes, and CD68 identified macrophages; CD20 provided no additional diagnostic information. No multinucleated giant cells were identified. The final report, validated approximately one month after the operation, diagnosed lymphocytic arteritis and advised exclusion of Horton's disease through clinical and biological correlation.

The report did not describe granulomas, an elastic stain, medial elastic fiber fragmentation, plasma-cell quantification, IgG4 immunostaining, or proximal/distal margin involvement. Original histological slides were unavailable for additional review. Accordingly, the specimen establishes lymphocytic aortitis but does not provide definitive histological confirmation of GCA or allow a more specific subtype to be assigned under the cardiovascular pathology consensus classification [12].

Postoperative course and follow-up

The early postoperative course was complicated by hemorrhagic shock requiring vasopressor support and

transfusion of one unit of packed red blood cells. Chest drainage reached approximately 1,520 mL over six hours, prompting surgical re-exploration on postoperative day one. Diffuse venous bleeding was controlled in the periaortic and retrosternal tissues; no single major arterial bleeding source was identified. Postoperative atrial fibrillation was treated with intravenous followed by oral amiodarone. The patient returned to sinus rhythm and was discharged home seven days after the primary operation.

The patient had already been treated with glucocorticoids for PMR before admission, although recorded outpatient hydrocortisone doses differed between contemporaneous notes (approximately 2.5-10 mg/day). During intensive care, hydrocortisone was administered intravenously at 50 mg four times daily. The discharge prescription specified oral hydrocortisone 40 mg each morning for two days. These prescriptions cannot be interpreted as evidence of a newly initiated, standardized high-dose GCA induction regimen. A complete GCA-specific starting dose and taper were not documented, and no tocilizumab treatment was documented.

FDG-PET/CT performed 47 days after the surgery, found no metabolic evidence of active large-vessel vasculitis. Moderate tracer uptake around the ascending aortic graft was interpreted as postoperative inflammation. The accompanying free-breathing, non-contrast CT showed postoperative changes, mild pericardial effusion, and bilateral pleural effusions, greater on the left. This CT component was not a dedicated contrast-enhanced angiogram and did not provide measurements or a formal assessment of the graft anastomoses.

At rheumatologic review approximately eight months after surgery, PMR was considered clinically in remission. Treatment comprised methotrexate 15 mg weekly and hydrocortisone 20 mg daily; intolerance to methylprednisolone, prednisone, and prednisolone was recorded. Approximately seven months after surgery, C-reactive protein was 9.15 mg/L and erythrocyte sedimentation rate 22 mm/h. The inflammatory markers could not be attributed exclusively to vasculitis because concurrent infection was also considered. Further laboratory and rheumatologic review was planned. Dedicated long-term angiographic surveillance of the graft and residual aorta remains indicated.

Discussion

This case illustrates three important issues for cardiac surgeons: the indication for urgent intervention in an exceptionally large and apparently rapidly enlarging ascending aortic aneurysm, the technical difficulty of operating in an inflammatory field, and the diagnostic value of routine histopathological examination of the resected aorta. The difference between approximately 40 mm at first check-up and 78 mm a year later is striking compared with published mean growth rates for nonsyndromic ascending aneurysms [9]. Nevertheless, as emphasized by Sonsino et al. [10], differences in imaging acquisition and measurement conventions may contribute to apparent growth. A directly standardized, same-level centerline comparison was not available here.

The focal ulceration increased concern for local aortic instability. Although the operative findings confirmed ulceration, the available CT report does not establish whether the lesion represented a classical atherosclerotic penetrating ulcer, inflammatory ulceration, or a focal pseudoaneurysmal component. Thus, the indication for expedited repair rested primarily on the extreme aneurysm diameter, ulcerated morphology, and clinical assessment of rupture risk, rather than proof of a specific acute aortic syndrome [11]. The operation followed hemodynamic stabilization and preoperative coronary assessment, supporting the designation of urgent rather than immediate emergency surgery.

Inflammatory changes also affected surgical technique. In the present operation, dissection of the ascending aorta was unusually difficult because inflammatory tissue obscured the normal tissue planes. Careful mobilization and meticulous handling were required before resection and graft replacement. The limited aortic root involvement and very mild regurgitation favored a supracoronary Dacron graft rather than root or valve replacement. Although separation from cardiopulmonary bypass was uncomplicated, postoperative bleeding required re-exploration; this morbidity should be considered when interpreting the surgical outcome.

The histopathological diagnosis was lymphocytic aortitis with inflammation across all three aortic wall layers. In a patient with PMR, this raises suspicion of GCA-associated aortic disease, but the absence of giant cells and the nonspecific lymphocytic pattern do not establish GCA [4,12]. Differential diagnoses include isolated noninfectious aortitis, other systemic inflammatory conditions, IgG4-related disease, and infectious aortitis. An extensive infectious workup, including bacterial, syphilitic, mycobacterial, viral, and fungal investigations, was negative, making an infectious etiology unlikely. Prior glucocorticoid exposure could have altered inflammatory activity, but it cannot be proven to explain the absence of giant cells.

The 2022 ACR/EULAR classification framework requires age ⩾50 years and at least six classification points [8]. Age was satisfied, but the documentation does not establish enough specific items to calculate a reliable qualifying score: PMR as a diagnosis is not itself a criterion, shoulder/neck morning stiffness was not characterized sufficiently, and the preoperative CRP was below the classification threshold. No temporal

artery biopsy or temporal/axillary ultrasound study was documented. The postoperative FDG-PET/CT did not show active large-vessel uptake, although prior aortic resection and ongoing immunomodulatory therapy limit retrospective interpretation. These criteria are designed for classification, not to exclude clinically suspected disease in an individual patient.

Aortic disease in GCA may remain asymptomatic, as illustrated in the series by Marie et al. [13], while Muratore et al. emphasized the relationship between PMR, large-vessel GCA, and vascular complications [5]. Strachan et al. described the prominence of ascending aortic aneurysms in GCA-associated thoracic aortic disease [14]. The relationship between inflammation and the apparent speed of aneurysmal enlargement in this patient remains biologically plausible but cannot be established causally from a single case.

Long-term follow-up requires collaboration between cardiac surgery, cardiology, and rheumatology. Current recommendations support assessment of inflammatory activity and structural vascular complications in suspected or established large-vessel GCA [6,7]. In this patient, the postoperative FDG-PET/CT addressed metabolic activity but its non-contrast CT component did not replace dedicated angiographic surveillance of the graft, anastomoses, or remaining aorta. A normal metabolic study after surgery should not be interpreted as proof that inflammatory aortitis was absent before resection.

Conclusions

An unusually large and rapidly enlarging ascending aortic aneurysm with focal ulceration may require urgent surgical treatment, particularly when an inflammatory aortopathy is suspected. This case demonstrates successful supracoronary replacement with a Dacron graft despite technically difficult dissection caused by surrounding inflammatory changes. Postoperative hemorrhage required re-exploration, followed by recovery and discharge home. Histopathological examination of the resected aorta revealed lymphocytic aortitis, raising suspicion of GCA in a patient with PMR without establishing a definitive causal diagnosis. Systematic examination of surgical specimens and long-term multidisciplinary surveillance are essential to recognize and manage potentially persistent aortic disease.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Skander HAFI, Jan Remes, Silvia Solari

Drafting of the manuscript: Skander HAFI, Silvia Solari

Critical review of the manuscript for important intellectual content: Jan Remes, Arnaud Henkens

Acquisition, analysis, or interpretation of data: Arnaud Henkens

Supervision: Silvia Solari

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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