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Ascending aortic intramural hematoma in a patient with granulomatosis with polyangiitis

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Granulomatosis with polyangiitis (GPA) is an antineutrophil cytoplasmic antibody–associated necrotizing vasculitis predominantly affecting small- and medium-sized vessels [1,2,3]. Although large-vessel involvement is rare, GPA may occasionally affect the aorta, leading to potentially life-threatening complications such as aneurysm formation, periaortitis, intramural hematoma, and dissection [1,2,4,5]. Cases of aortic intramural hematoma associated with GPA

remain exceptionally uncommon and may progress to rupture [1]. Due to their nonspecific clinical presentation, these manifestations may remain underrecognized. To the best of our knowledge, we present the first reported case of GPA-associated intramural hematoma involving the ascending aorta detected on computed tomography (CT).

A 68-year-old man was admitted to the Department of Rheumatology and Immunology in January 2026 with suspected GPA based on recurrent epistaxis, proteinuria, progressive renal dysfunction, and anemia. His medical history included heart failure, arterial hypertension, chronic kidney disease, and chronic superficial gastritis. Hypertension was well controlled with antihypertensive therapy. The patient denied chronic smoking, and the family history was unremarkable. No chest trauma had occurred prior to admission.

The patient reported recurrent epistaxis for approximately five years, which intensified after the initiation of antiplatelet therapy following ST-segment elevation myocardial infarction in October 2025. The antithrombotic regimen consisted of aspirin 75 mg once daily and clopidogrel 75 mg once daily. During the two weeks preceding admission, he experienced marked fatigue, generalized weakness, and an unintentional weight loss of approximately 10 kg. On admission, the patient was in good general condition and remained hemodynamically and respiratorily stable. Physical examination revealed deformities of the finger joints and nasal speech. Laboratory tests showed moderate microcytic anemia, mild lymphopenia, and renal dysfunction (serum creatinine 217 μmol/L; reference range [RR] 53–115 μmol/L; estimated glomerular filtration rate 27 ml/min/1.73 m²; RR ≥ 90 ml/min/1,73 m²). Inflammatory markers were elevated (C-reactive protein 21.6 mg/L; RR < 5 mg/L). Urinalysis revealed proteinuria, hematuria, and glucosuria, likely related to dapagliflozin therapy, with protein excretion of 1.33 g/24 h (RR < 0.15 g/24 h). Immunological testing demonstrated positive anti-proteinase 3 antibodies (anti-proteinase 3 antibody 59.0 IU/mL; RR < 2.0 IU/mL). High-resolution computed tomography (CT) of the chest revealed multiple irregular spiculated pulmonary

nodules without cavitation and adjacent ground-glass opacities suggestive of possible alveolar hemorrhage (Figure 1A). Additionally, CT demonstrated thickening of the ascending aortic wall consistent with an intramural hematoma (Figure 1B, 1C, and 1D). The thickened wall measured up to approximately 8 mm, appeared hyperdense on non-contrast imaging (57 Hounsfield units), and showed no significant post-contrast enhancement, consistent with intramural hematoma, likely associated with GPA. Bronchoscopy with bronchoalveolar lavage, nasal mucosa biopsy, and kidney biopsy were recommended but declined by the patient. Treatment with systemic glucocorticoids and rituximab was initiated, and the patient was scheduled for further cardiology follow-up.

Large-vessel involvement in GPA is uncommon but clinically significant, as delayed recognition may lead to severe complications, including aortic dissection or rupture [1,2,4]. Management typically includes immunosuppressive therapy and close cardiovascular surveillance, while selected patients may require surgical or endovascular intervention. Prognosis largely depends on the extent of aortic involvement and the timelines of diagnosis and treatment. [1,2,5,6]. In addition to autoimmune inflammation, established risk factors for aortic disease, including poorly controlled hypertension, smoking, a family history of aortic aneurysm or dissection, and atherosclerotic disease, should be considered when evaluating patients with the suspected large-vessel involvement in GPA. Given the nonspecific clinical presentation and the potential for life-threatening complications, early imaging is essential in the assessment of these patients. CT angiography plays a key role in detecting aortic abnormalities, including intramural hematoma, aneurysm, and periaortic inflammation. In patients with GPA—especially those presenting with atypical symptoms, persistent inflammation, or new-onset chest pain- CT imaging should be considered to exclude large-vessel involvement and facilitate timely management [1,4,6].

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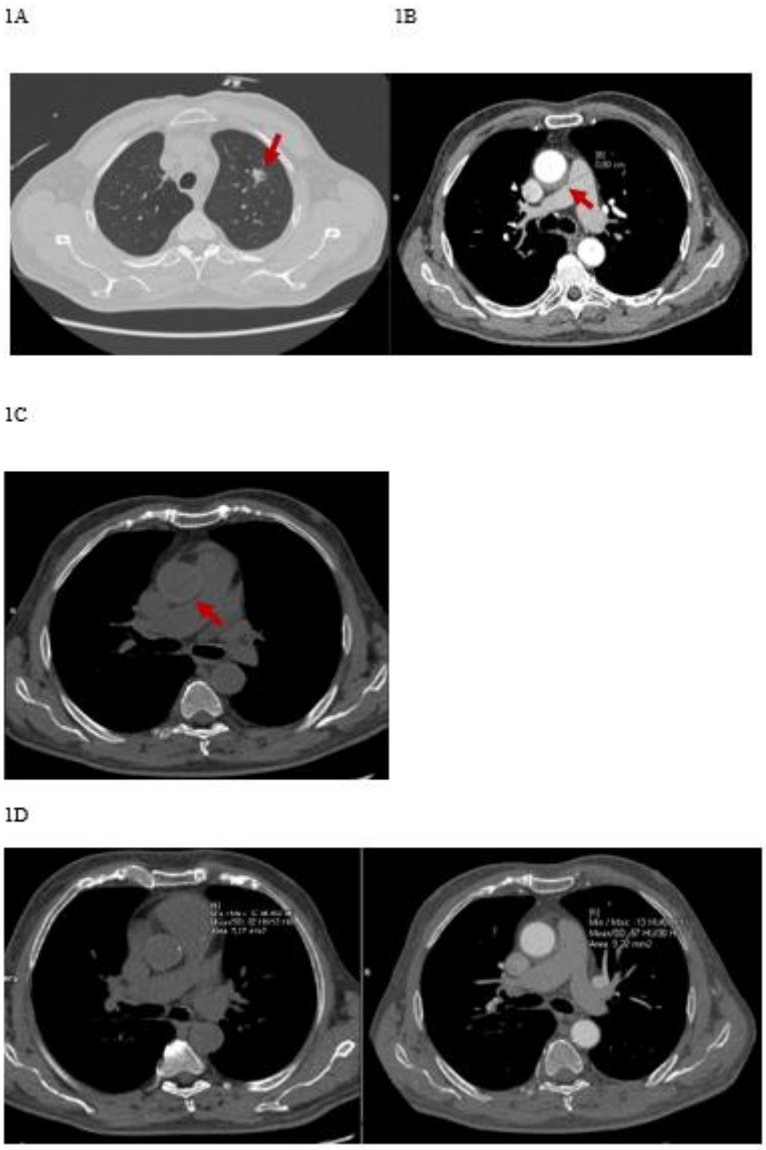

Figure 1 A – axial chest computed tomography (CT) demonstrating multiple irregular spiculated pulmonary nodules (arrow); **B** – contrast-enhanced axial CT image showing focal thickening of the posterior wall of the ascending aorta (arrow), measuring approximately 8 mm, consistent with an aortic intramural hematoma; **C** – axial noncontrast CT of the thorax demonstrating hyperdense thickening of the ascending aortic wall (arrow), consistent with an

intramural hematoma; **D** – axial CT images obtained before (left) and after (right) contrast administration demonstrating thickening of the posterior wall of the ascending aorta, without significant contrast enhancement, consistent with an intramural hematoma

Short title: Ascending aortic intramural hematoma in a patient with GPA