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A 78-Year-Old Man With a Previously Stable Atherosclerotic Abdominal Aortic Aneurysm Presenting With Impending Rupture Following COVID-19

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None declared

Patient: Male, 78-year-old**Final Diagnosis:** Impending rupture of abdominal aortic aneurysm**Symptoms:** Recurrent fever • elevated inflammatory markers • mild low back pain**Clinical Procedure:** —**Specialty:** Cardiac Surgery**Objective:** Unusual clinical course**Background:** Abdominal aortic aneurysm (AAA) rupture is a life-threatening condition. Although elective repair is generally guided by aneurysm size, impending rupture can occur when rapid aneurysmal expansion or periaortic inflammatory changes develop before overt hemodynamic deterioration. This report describes the case of a 78-year-old man with a previously stable 43-mm atherosclerotic AAA who presented with impending rupture after coronavirus disease 2019 (COVID-19).**Case Report:** A 78-year-old Japanese man with a known AAA under imaging surveillance developed fever and was diagnosed with COVID-19 by nasopharyngeal polymerase chain reaction testing. Seven days later, he was admitted for recurrent fever, cough, poor appetite, and elevated inflammatory markers. His medical history included hypertension, heavy smoking, advanced chronic kidney disease, and diffuse atherosclerotic disease. The aneurysm had shown minimal growth over 2 years, with a stable maximum short-axis diameter of approximately 43 mm on serial computed tomography. After admission, C-reactive protein levels initially decreased but rose again on hospital day 11, accompanied by new-onset mild low back pain and no marked blood pressure elevation or hemodynamic instability. Repeat non-contrast abdominal computed tomography performed 18 days after COVID-19 diagnosis demonstrated rapid AAA expansion from 43 mm to 51 mm within 2 months, with periaortic fat stranding, consistent with impending rupture. Urgent endovascular aneurysm repair was performed before hemodynamic deterioration. The postoperative course was stable, and the patient was discharged without ischemic or procedural complications.**Conclusions:** This case shows that a previously stable AAA can abruptly destabilize after recent COVID-19. Repeat imaging should be considered when inflammatory markers rise or new symptoms occur, even when symptoms are mild.**Keywords:** Aneurysm • Cardiovascular Diseases • Case Reports • COVID-19 • Endovascular Aneurysm Repair • InflammationFull-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/954216> 1700 — 2 16

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Introduction

Abdominal aortic aneurysm (AAA) is most commonly encountered in older men, particularly those with a history of smoking, and population-based screening studies have reported AAA prevalence ranging from 1.6% to 7.2% among adults aged 60 to 65 years or older [1]. Atherosclerotic AAA is often asymptomatic, but symptomatic patients can present with abdominal, back, or flank pain; diagnosis is commonly established by ultrasonography or computed tomography, and management is guided by aneurysm size, growth rate, symptoms, and rupture risk [2,3]. AAA carries a substantial risk of fatal rupture once the aneurysm diameter exceeds 50-55 mm [2,3]. Impending rupture represents a critical pre-rupture state characterized by rapid aneurysmal expansion or periaortic inflammatory changes, often in the absence of overt hemodynamic instability [3]. Systemic inflammation, endothelial dysfunction, and thrombo-inflammatory responses associated with viral infection, including coronavirus disease 2019 (COVID-19), can contribute to aneurysm instability in susceptible patients [4,5]. Previous case reports have described AAA rupture during active or recent COVID-19 illness, although evidence remains limited [5,6]. Patients can present with minimal or nonspecific symptoms, making diagnosis challenging [7]; therefore, current guidelines

emphasize temporal changes on serial imaging as central to recognizing aneurysmal instability. Early identification of this high-risk state allows timely endovascular repair and can improve clinical outcomes [3]. This report describes the case of a 78-year-old man with a previously stable 43-mm atherosclerotic AAA who developed imaging findings consistent with impending rupture at 51 mm, 18 days after COVID-19 diagnosis, and was managed with urgent endovascular aneurysm repair.

Case Report

A 78-year-old Japanese man with a known abdominal aortic aneurysm (AAA) was admitted for recurrent fever and elevated inflammatory markers following coronavirus disease 2019 (COVID-19). The AAA had been detected several years earlier and had shown slow, stable enlargement during routine imaging follow-up. Serial computed tomography (CT) showed a stable maximum short-axis diameter of the AAA, remained stable at approximately 43 mm over 2 years (Figure 1A). Seven days before admission, the patient developed fever and was diagnosed with COVID-19 by nasopharyngeal polymerase chain reaction (PCR) testing. SARS-CoV-2 variant information and COVID-19 vaccination status were not available in the clinical

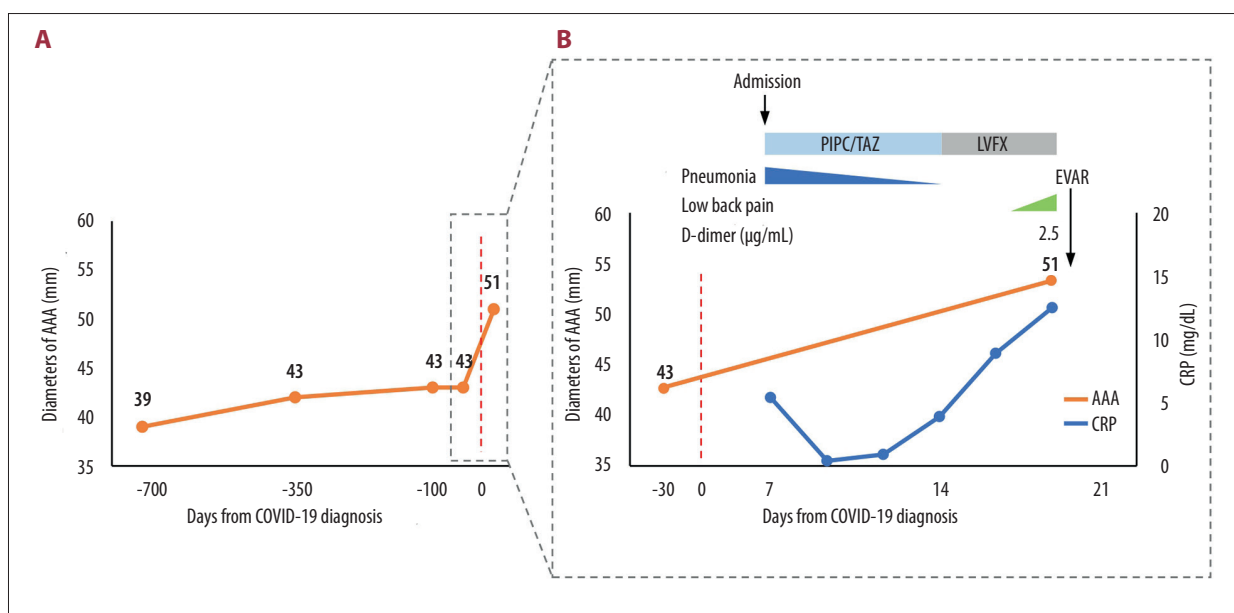


Figure 1. Long-term abdominal aortic aneurysm growth and short-term clinical course after COVID-19 diagnosis. (A) Long-term changes in abdominal aortic aneurysm (AAA) diameter plotted against days from coronavirus disease 2019 (COVID-19) diagnosis. The aneurysm had demonstrated slow, gradual enlargement over approximately 2 years, followed by rapid enlargement from 43 mm to 51 mm after COVID-19. The dashed red line indicates the timing of COVID-19 diagnosis. AAA diameter was defined as the maximal short-axis diameter on computed tomography. (B) Magnified view of the short-term clinical course after COVID-19 diagnosis. Changes in AAA diameter with serum C-reactive protein (CRP) levels, timing of hospital admission, antibiotic administration, pneumonia, low back pain, D-dimer level, and endovascular aneurysm repair (EVAR). After an initial CRP decrease with antibiotic therapy, CRP rose again, accompanied by new-onset low back pain and rapid AAA enlargement, leading to the diagnosis of impending AAA rupture and subsequent EVAR. Shaded bars indicate the administration periods of piperacillin/tazobactam (PIPC/TAZ) and levofloxacin (LVFX).

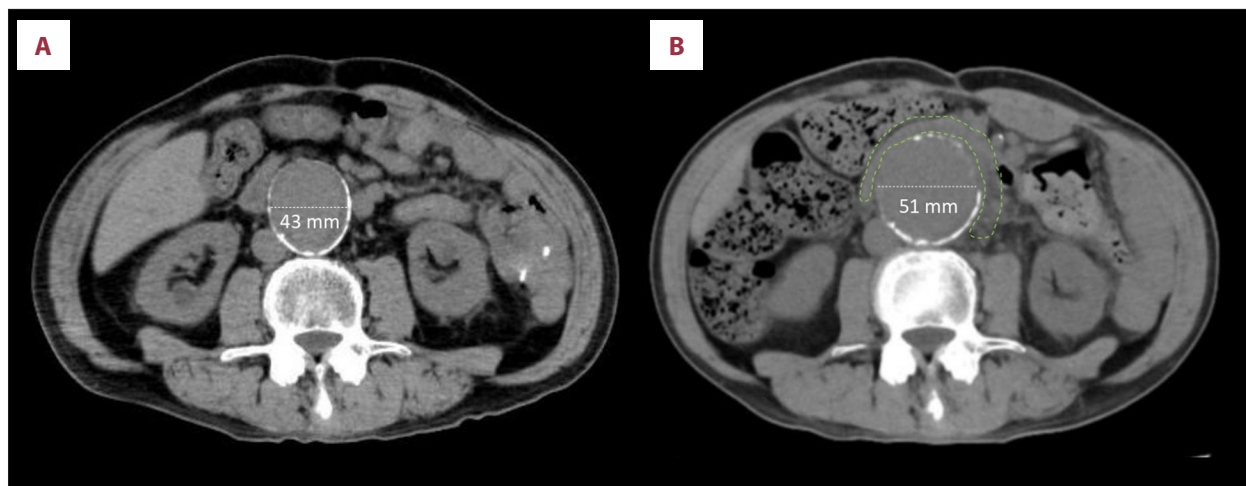


Figure 2. Serial non-contrast computed tomography images showing rapid abdominal aortic aneurysm enlargement after COVID-19 diagnosis. (A) Axial non-contrast computed tomography image obtained 35 days before coronavirus disease 2019 (COVID-19) diagnosis, demonstrating an abdominal aortic aneurysm (AAA) measuring 43 mm in maximal short-axis diameter. (B) Axial non-contrast computed tomography image obtained 18 days after COVID-19 diagnosis, showing rapid aneurysm enlargement to 51 mm in maximal short-axis diameter, accompanied by increased periaortic fat attenuation (highlighted by the light green dashed outline), consistent with impending AAA rupture.

record. Oral molnupiravir was prescribed, resulting in transient defervescence. However, several days later, fever recurred with cough and poor appetite, prompting re-evaluation and hospital admission. His medical history included hypertension, type 2 diabetes mellitus, peripheral artery disease, ischemic stroke, and advanced chronic kidney disease (stage 5) attributed to diabetic kidney disease. Blood pressure and volume status had been chronically elevated but stable under optimized management. He had a long smoking history.

On admission (7 days after COVID-19 diagnosis), his temperature was 38.1 °C, with pulse rate 63 beats/min and blood pressure 157/74 mm Hg. Chest CT revealed mild bilateral ground-glass opacities and small pleural effusions. Laboratory testing revealed an elevated C-reactive protein (CRP) level of 5.4 mg/dL and a serum creatinine level of 4.9 mg/dL, corresponding to an estimated glomerular filtration rate of 9.8 mL/min/1.73 m², according to the Japanese Society of Nephrology equation [8].

Given his persistent fever and pulmonary findings on chest CT, empiric broad-spectrum antibiotics, including piperacillin–tazobactam, were initiated along with diuretic therapy. His blood pressure remained stable and comparable to pre-admission outpatient levels throughout this period. Despite improvement in respiratory status and an initial decrease in CRP levels to 0.4 mg/dL by hospital day 3, CRP levels subsequently rose again, reaching 12.5 mg/dL on hospital day 11 (Figure 1B). At the time of CRP re-elevation, respiratory symptoms and oxygenation had improved, and no definite alternative source of inflammation was identified clinically. On the same day, 18 days after COVID-19 diagnosis, he reported new-onset mild low

back pain. Non-contrast abdominal CT demonstrated rapid enlargement of the AAA from 43 mm to 51 mm within 2 months (Figure 2A, 2B), accompanied by periaortic fat stranding (Figure 2B) and a D-dimer level of 2.3 µg/mL, findings consistent with impending aneurysm rupture. Given the acute change from a previously stable growth pattern, urgent endovascular aneurysm repair (EVAR) was performed on hospital day 12.

The postoperative course was clinically stable. Hemodialysis was initiated on hospital day 14 to manage postoperative fluid balance and uremia. Blood pressure stabilized at approximately 120/60 mm Hg, inflammatory markers normalized, and low back pain resolved completely, with no ischemic or procedural complications. The patient was discharged in stable condition and transitioned to outpatient hemodialysis care.

Discussion

This case report demonstrates that a previously stable AAA can abruptly destabilize with only mild symptoms, and that repeat imaging is important when inflammatory markers rise or new symptoms develop. Although AAAs are typically managed based on size thresholds, some lesions undergo abrupt destabilization before reaching the conventional criteria for intervention. Impending rupture is a clinically important state marked by rapid expansion or periaortic inflammatory changes, often without overt hemodynamic compromise [3]. In such cases, subtle symptoms and dynamic changes on serial imaging may provide the only clues to evolving instability and should prompt timely reassessment and intervention [7].

In the present case, the patient had a known AAA and multiple established risk factors for aneurysmal progression, including long-standing hypertension, CKD, a heavy smoking history, advanced age, and extensive atherosclerotic disease. The aneurysm had demonstrated minimal growth over a 2-year period, with an increase of only 4 mm in the maximum short-axis diameter, before undergoing abrupt expansion of 8 mm in the maximum short-axis diameter within the final 2 months, accompanied by periaortic fat stranding. Notably, this rapid change was observed in close temporal proximity to a recent COVID-19 diagnosis, approximately 7 days prior to hospital admission. This marked deviation from the expected growth pattern is consistent with abrupt aneurysmal destabilization rather than gradual chronic progression, underscoring the clinical importance of reassessing aneurysm stability when the disease course deviates from prior behavior.

Systemic inflammation is an established contributor to AAA pathogenesis and progression. Experimental and clinical studies have shown that inflammatory cytokines, immune cell infiltration, and matrix metalloproteinase activation promote degradation of the aortic wall, leading to aneurysmal expansion and rupture. Elevated CRP levels have been associated with both aneurysm expansion and inflammatory activity within the aneurysm wall [9]. COVID-19 has been reported to involve the cardiovascular system and to increase the risk of vascular complications. Large population-based studies have further demonstrated an increased risk of acute vascular events, including myocardial infarction and ischemic stroke, following COVID-19 [10]. Mechanistically, SARS-CoV-2 infection induces endothelial dysfunction, platelet activation, and immunothrombotic responses, resulting in a prothrombotic and vasculopathic milieu [11]. Histopathological evidence of endotheliitis has further supported the concept that SARS-CoV-2 directly affects the vascular endothelium [12]. Importantly, emerging evidence suggests that endothelial dysfunction and prothrombotic alterations can persist beyond the acute phase of COVID-19, potentially extending the period of vascular vulnerability [13]. These observations provide biological plausibility for vascular vulnerability after COVID-19, but they do not establish a causal relationship between COVID-19 and aneurysmal destabilization in an individual patient.

Although a direct causal relationship cannot be established from a single case, the temporal proximity between COVID-19 infection and abrupt aneurysmal destabilization in this patient is clinically compelling. Importantly, no marked blood pressure elevation or hemodynamic instability was observed before or after admission, suggesting that the rapid aneurysmal expansion was unlikely to have been driven by acute hypertensive stress alone. In addition, histopathological evaluation of the aneurysm wall was not available because EVAR was performed; nevertheless, the abrupt deviation from a long-standing stable

growth pattern is more consistent with an acute destabilizing process than with gradual chronic progression. Pre-existing AAA represents a structurally compromised vascular segment that can be vulnerable to systemic inflammatory or endothelial perturbations. In this context, recent COVID-19 should be interpreted as a clinically relevant temporal context rather than as a proven causal trigger of aneurysmal instability. Previous reports have described AAA rupture during active or recent COVID-19, including cases treated with endovascular or open surgical repair [5,6]. Rupture of a small, sub-threshold AAA has also been reported, suggesting that size alone may not fully capture rupture risk [14]. In contrast, the present case was diagnosed before hemodynamic deterioration as an imaging-defined impending rupture, based on rapid interval enlargement on CT and periaortic fat stranding.

The value of this case report lies not in establishing COVID-19 as a cause of AAA destabilization, but in illustrating a clinically important pattern: a previously stable, sub-threshold AAA underwent rapid enlargement with imaging features of impending rupture in close temporal association with recent COVID-19. This case also emphasizes that symptom severity alone may be insufficient to detect aneurysmal instability and that repeat imaging can be decisive when inflammatory markers rise or new symptoms occur.

Patients with advanced CKD are a vulnerable population with respect to aortic aneurysm-related complications. CKD is associated with endothelial dysfunction, vascular calcification, and accelerated atherosclerosis [15], all of which can compromise arterial wall integrity. Outcomes after AAA rupture or repair are known to be substantially worse in patients with advanced CKD or end-stage kidney disease than in those with preserved renal function [16]. In this case, early recognition of aneurysmal instability enabled timely intervention and a favorable outcome despite significant comorbidity.

This case report shows that abrupt aneurysmal destabilization can occur even after a prolonged period of stable growth. Temporal changes on serial imaging, rather than symptom severity alone, were critical for identifying impending rupture and guiding timely repair. Recent COVID-19 should therefore be viewed as a clinical context that may warrant reassessment of known AAA when inflammatory markers rise or new symptoms occur, rather than as evidence of a causal mechanism.

Conclusions

This case shows that a previously stable AAA can abruptly destabilize after recent COVID-19. Careful reassessment with serial imaging should be considered when inflammatory markers rise or new symptoms occur, as it may help identify

impending rupture and enable timely intervention even when symptoms are mild.

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Declaration About Informed Consent

Consent for publication was obtained.

AI Disclosure

GPT-5.4 (OpenAI) was used for English language editing and grammatical refinement.

Declaration of Figures' Authenticity

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