

Point-of-Care Ultrasound in the Diagnosis of Extensive Stanford Type A Aortic Dissection in a Low-Resource Setting: a case report

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Case Report

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Abstract

Background

Acute aortic dissection (AAD) carries high early mortality and is frequently misdiagnosed, particularly in resource-limited settings where computed tomography angiography (CTA) is inaccessible and atypical presentations are common. Point-of-care ultrasound (POCUS) has emerged as a critical first-line diagnostic adjunct, yet documented experience from West African emergency departments is virtually absent. To our knowledge, this is the first reported case of emergency physician-performed POCUS leading to the diagnosis of aortic dissection in a Ghanaian emergency department.

Case presentation:

A 74-year-old Ghanaian man with newly diagnosed hypertension presented to the emergency department (ED) of Komfo Anokye Teaching Hospital (KATH), Kumasi, Ghana, with a two-week history of atypical central chest pain, palpitations, and exertional fatigue – without the classical abrupt tearing onset. Bedside POCUS was performed using the Sonographic Protocol for the Emergent Evaluation of Aortic Dissections (SPEED) framework, incorporating parasternal, subxiphoid, and abdominal aortic windows. POCUS demonstrated marked aortic dilatation (5.21–6.35 cm across multiple views) and a visible mobile intimal flap with true and false lumen separation. CTA confirmed extensive Stanford type A (DeBakey I) aortic dissection from the aortic root through the thoracoabdominal aorta into the right common iliac artery, with giant aneurysmal dilatation (aortic root 80 mm) and branch-vessel involvement. A mildly elevated troponin I (0.08 ng/mL) was correctly attributed to aortic root involvement rather than primary acute coronary syndrome, based on a normal ECG and overall clinical context. Definitive open surgical repair was not feasible due to local infrastructure limitations. Anti-impulse therapy was initiated and the patient was discharged haemodynamically stable after 12 days, remaining well at 30-day follow-up.

Conclusions

This case demonstrates that POCUS using the SPEED protocol can be successfully applied in a resource-limited West African ED to rapidly identify life-threatening aortic pathology with an atypical presentation. It highlights a critical diagnostic-therapeutic gap: the tools to identify extensive AAD are achievable with POCUS, but the surgical infrastructure to treat it remains severely limited across sub-Saharan Africa. Expanding emergency POCUS training in the region is an achievable near-term priority.

Background

Acute aortic dissection (AAD) is a life-threatening cardiovascular emergency arising from an intimal tear that propagates blood into the aortic media, creating true and false lumina. Stanford type A dissections

(involving the ascending aorta) require emergency surgery and carry an approximate 1–2% hourly mortality if untreated, with 30-day mortality approaching 50% without intervention [1, 2]. Globally, AAD occurs at an incidence of 2.9–3.5 per 100,000 person-years, predominantly affecting older males [3]. In sub-Saharan Africa, robust epidemiological data are virtually absent; a systematic review identified fewer than 200 documented cases across Africa over 20 years, almost certainly reflecting profound underdiagnosis rather than a genuinely lower burden [4]. Hypertension – the dominant modifiable risk factor for AAD worldwide – is highly prevalent and frequently poorly controlled in the region, where fewer than one in five hypertensive individuals achieves adequate blood pressure control [5].

Diagnosis of AAD is further complicated by its protean presentations. While abrupt tearing pain is the classical feature, up to 20% of patients present without it, and atypical features are common [6]. In African studies, diagnostic delays exceeding 48 hours and misdiagnosis rates as high as 38% are documented [4]. CTA remains the gold standard (sensitivity and specificity exceeding 95%) [7], but access is severely restricted across much of sub-Saharan Africa. POCUS has therefore emerged as a critical diagnostic adjunct: the SPEED protocol achieves sensitivity of 93.2%, specificity of 90.9%, and negative predictive value of 99.7% [8], and focused cardiac ultrasound has been shown to reduce median time to diagnosis from 226 to 80 minutes [9].

We present a case of extensive Stanford type A aortic dissection in a 74-year-old Ghanaian man with an atypical clinical presentation, diagnosed by bedside POCUS in the emergency department of KATH – the first such documented case in Ghana.

Case Presentation

A 74-year-old Ghanaian man presented to the emergency department of Komfo Anokye Teaching Hospital (KATH), Kumasi, Ghana – a national tertiary referral center – with a two-week history of mild intermittent central non-radiating chest pain (pain score 4/10), palpitations, and progressive exertional fatigue. He denied diaphoresis, syncope, abdominal or back pain, limb weakness, or other neurological symptoms. He had been recently diagnosed with hypertension at a peripheral health facility and started on amlodipine 5 mg daily, prescribed about four weeks prior to presentation. He reported a history of social alcohol use and cigarette smoking of approximately 10 pack-years, with no other known chronic illnesses.

On arrival, he was conscious, alert and self-ambulating. Vital signs: blood pressure 160/91 mmHg (right arm), with no significant variation in the left arm, heart rate 90 bpm, respiratory rate 18 cycles/min, SpO₂ 97% on room air, temperature 36.7°C, random blood glucose 6.2 mmol/L. Blood pressure was equal in both arms with no radio-radial or radio-femoral delay. Heart sounds were normal with no audible murmur. Respiratory and abdominal examinations were unremarkable. Neurological examination was grossly normal.

Investigations

A 12-lead ECG demonstrated sinus rhythm at 67 bpm with no significant abnormalities. Neither the Cornell voltage criteria (R in aVL + S in V3 ≥ 2.8 mV in males) nor the Sokolow-Lyon criterion ($RV5 + SV1 \geq 3.5$ mV) were met. No ST-segment changes, ischaemic T-wave inversions, conduction abnormalities, or voltage criteria for left ventricular hypertrophy were identified (Fig. 1). The ECG impression was normal sinus rhythm.

Laboratory investigations

Serum troponin I was mildly elevated at 0.08 ng/mL (reference < 0.04 ng/mL). In the absence of ST-segment changes or a typical ischemic symptom profile, this was interpreted as myocardial stress secondary to aortic root involvement and hemodynamic strain – a recognized pattern in aortic dissection with coronary ostial involvement – rather than a primary Type 1 myocardial infarction [10]. Full blood count demonstrated mild normocytic normochromic anemia (haemoglobin 10.8 g/dL, MCV 86 fL), without reticulocytosis, consistent with anaemia of chronic disease. Renal function (urea 4.2 mmol/L, creatinine 88 μ mol/L, eGFR > 60 mL/min/1.73 m²), serum electrolytes, fasting lipid profile, and coagulation studies were within normal limits. Liver enzymes showed mild AST elevation at 1.8 $\times$ the upper limit of normal with normal synthetic function, suggesting mild hepatic congestion.

Point-of-Care Ultrasound

POCUS was performed by emergency physicians using the SPEED protocol framework. The examination incorporated parasternal long-axis (PLAX), parasternal short-axis (PSAX), apical four-chamber, and subxiphoid cardiac windows, followed by transverse and longitudinal abdominal aortic views and a FAST examination.

Cardiac POCUS showed no pericardial effusion. The aortic root was markedly dilated at 5.21 cm (measured at 2 cm from the aortic valve annulus; normal < 3.7 cm) on the PLAX view (Fig. 2). The abdominal aorta was similarly dilated at 5.37 cm on longitudinal view (Fig. 1). The proximal abdominal aorta on short-axis subxiphoid view demonstrated a grossly dilated lumen of 5.21 cm with a visible mobile intimal flap (Figs. 2, 3). No pleural effusion or free intraperitoneal fluid was identified.

Based on these findings, a provisional diagnosis of Stanford type A aortic dissection was made. An urgent CT aortogram was ordered.

CT Angiography

CTA was performed at an external facility (the in-house CT scanner was temporarily non-operational). Imaging demonstrated extensive Stanford type A (DeBakey I) aortic dissection with patent true and false

lumina, extending from the aortic root and ascending aorta through the arch, descending thoracic aorta, and abdominal aorta into the right common iliac artery (Fig. 6).

Giant aneurysmal dilatation was documented: root 80 mm, ascending aorta 65 mm, aortic arch 70 mm, descending thoracic aorta 73 mm, abdominal aorta 53 mm. An intramural hematoma within the thoracic aorta was identified (length 10.9 cm, maximal thickness 2.0 cm). Branch-vessel involvement included the coeliac trunk, superior mesenteric artery, and left renal artery arising from the false lumen, with the right renal artery from the true lumen. An incidental aberrant right subclavian artery (arteria lusoria) was noted.

Management and Outcome

Cardiothoracic surgery consultation was obtained at KATH and multiple regional tertiary centers. Definitive open surgical repair could not be offered locally owing to lesion complexity and limitations in operative infrastructure; transfer to a higher-level center was recommended but logistically not feasible. Anti-impulse therapy was initiated immediately with intravenous beta-blockade (labetalol boluses), targeting a systolic blood pressure of < 110 mmHg and heart rate < 60 bpm. The patient was subsequently managed conservatively with continued anti-impulse therapy. Oral antihypertensive therapy was optimised with a calcium channel blocker, an angiotensin receptor blocker, and a beta-blocker. Formal echocardiography demonstrated aortic regurgitation without haemodynamically significant tamponade. The patient remained haemodynamically stable throughout an inpatient admission of 12 days and was discharged on beta-blocker therapy with cardiothoracic outpatient follow-up. At 30-day follow-up, he remained ambulant with stable vital signs without surgical intervention, managed medically as a high-risk operative candidate given the extent of disease.

Discussion

To our knowledge, no prior published case of emergency physician-performed POCUS leading to the diagnosis of aortic dissection has been documented from Ghana or any West African emergency department. The broader African literature on AAD is sparse and consists predominantly of retrospective case series and small cohort studies from North and South Africa; a comprehensive systematic review identified fewer than 200 documented cases across the entire continent over a 20-year period [4]. Published case reports describing POCUS-facilitated diagnosis of aortic dissection have almost exclusively originated from high-income settings in North America and Europe [15]. The present case therefore represents a significant geographic first and makes a direct contribution to the nascent body of literature on emergency cardiovascular imaging in sub-Saharan West Africa.

This case illustrates several clinically important themes in the diagnosis and management of AAD in resource-limited African settings. The patient – an elderly Ghanaian male with hypertension and a

smoking history — represents the archetypal demographic at risk [3, 6]. Ghana's hypertension prevalence exceeds 30% among adults, with awareness and control rates remaining low [11]; given that hypertension accounts for approximately 70–80% of all aortic dissections [1], and in African cohorts it is the most consistently uncontrolled risk factor at presentation [4, 12], the true burden of AAD in Ghana is almost certainly substantially higher than documented data suggest.

The atypical presentation — two weeks of mild chest discomfort without abrupt onset — is a recognised pattern in older patients with pre-existing aneurysmal dilatation [16]. The giant aortic root dilatation (80 mm) in this patient likely contributed to an attenuated pain response through progressive rather than acute intimal disruption [17]. This underscores the importance of routinely measuring the aortic root and abdominal aorta during ED cardiac POCUS in any patient with cardiovascular risk factors and unexplained cardiorespiratory symptoms.

The mildly elevated troponin I (0.08 ng/mL) merits specific emphasis as a common source of diagnostic error. In AAD involving the ascending aorta, troponin rise can reflect coronary ostial involvement, haemodynamic compromise, or non-ischaemic myocardial stress [10]. Misattributing this to primary ACS and initiating anticoagulation or thrombolysis without excluding dissection carries potentially fatal consequences. In this case, the normal ECG, sub-threshold elevation, two-week symptom trajectory, and overall clinical context appropriately shifted pre-test probability toward aortic pathology and prompted POCUS evaluation. Troponin elevation must never be used to exclude AAD in a clinically consistent presentation.

POCUS using the SPEED protocol provided two diagnostically compelling findings: aortic dilatation across multiple views (5.21–6.35 cm) and a visible mobile intimal flap with true and false lumen separation. Direct flap visualisation by emergency physician POCUS carries high specificity, though sensitivity varies [15]; a positive scan, as here, appropriately escalated care and enabled CTA to be secured before the in-house scanner was available. An underappreciated additional role of POCUS in resource-limited settings is as a communication tool: bedside cineloops demonstrating an intimal flap are far more persuasive in referral negotiations than a verbal clinical description, and can directly accelerate acceptance at a receiving centre.

Even with timely POCUS-based diagnosis and CTA confirmation, definitive surgical repair was inaccessible. Open repair for extensive type A dissection requires cardiopulmonary bypass, deep hypothermic circulatory arrest, and experienced aortic surgical teams — capabilities limited across sub-Saharan Africa [4, 18]. This diagnostic-therapeutic gap is a critical systems-level challenge: the tools to identify the disease now exist, but the capacity to treat it does not. Addressing this requires investment in regional surgical infrastructure and referral networks. In the interim, aggressive anti-impulse therapy remains the cornerstone of management for surgically ineligible patients. At the diagnostic level, expanding POCUS training through established frameworks such as the West African College of Physicians emergency ultrasound curriculum is an achievable, cost-effective near-term intervention that could meaningfully reduce diagnostic delay across Ghana and West Africa.

Conclusions

We present the first documented case of emergency physician-performed POCUS leading to the diagnosis of acute aortic dissection in a Ghanaian ED. The case demonstrates that the SPEED protocol can be successfully applied in a resource-limited West African setting to rapidly identify life-threatening aortic pathology with atypical presentation. It further highlights a persistent diagnostic-therapeutic gap: timely diagnosis is now achievable with POCUS, but the surgical infrastructure to treat extensive type A dissection remains critically limited. Expanding emergency POCUS training across West Africa is an achievable near-term priority; addressing the surgical gap requires sustained systemic investment. POCUS is not a substitute for CTA, but in the emergency evaluation of suspected acute aortic syndrome where advanced imaging is inaccessible, it is an indispensable and potentially life-saving first step.

Declarations

Competing interests: The authors declare that they have no competing interests."

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Authors' contributions: **Frederick Asamoah-Boachie**, *M.D., MSc.OEHS, CDM* (Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Visualization; Writing – original draft; Writing – review & editing), **Dr. Nessya Taylor**, *MGCS* (Resources; Supervision; Writing – review & editing), **Dr. Kwabena Danso**, *MGCS* (Resources; Supervision; Writing – review & editing), **Dr. Emmanuel Amoah**, *MBChB* (Investigation; Writing – review & editing).

Availability of data and materials: All data supporting the conclusions of this article are included within the article and its supplementary files. De-identified clinical data are available from the corresponding author on reasonable request.

Ethics approval and consent to participate: This case report was conducted in accordance with the Declaration of Helsinki. Institutional review board exemption was granted for de-identified case material.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the signed consent form is available for review by the Editor-in-Chief of this journal upon request.

Reporting guidelines: This case report was prepared in accordance with the CARE (CAse REport) guidelines. A completed CARE checklist is provided as Additional file 1.

Use of large language models: : No large language models were used in the preparation of this manuscript.

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Figures

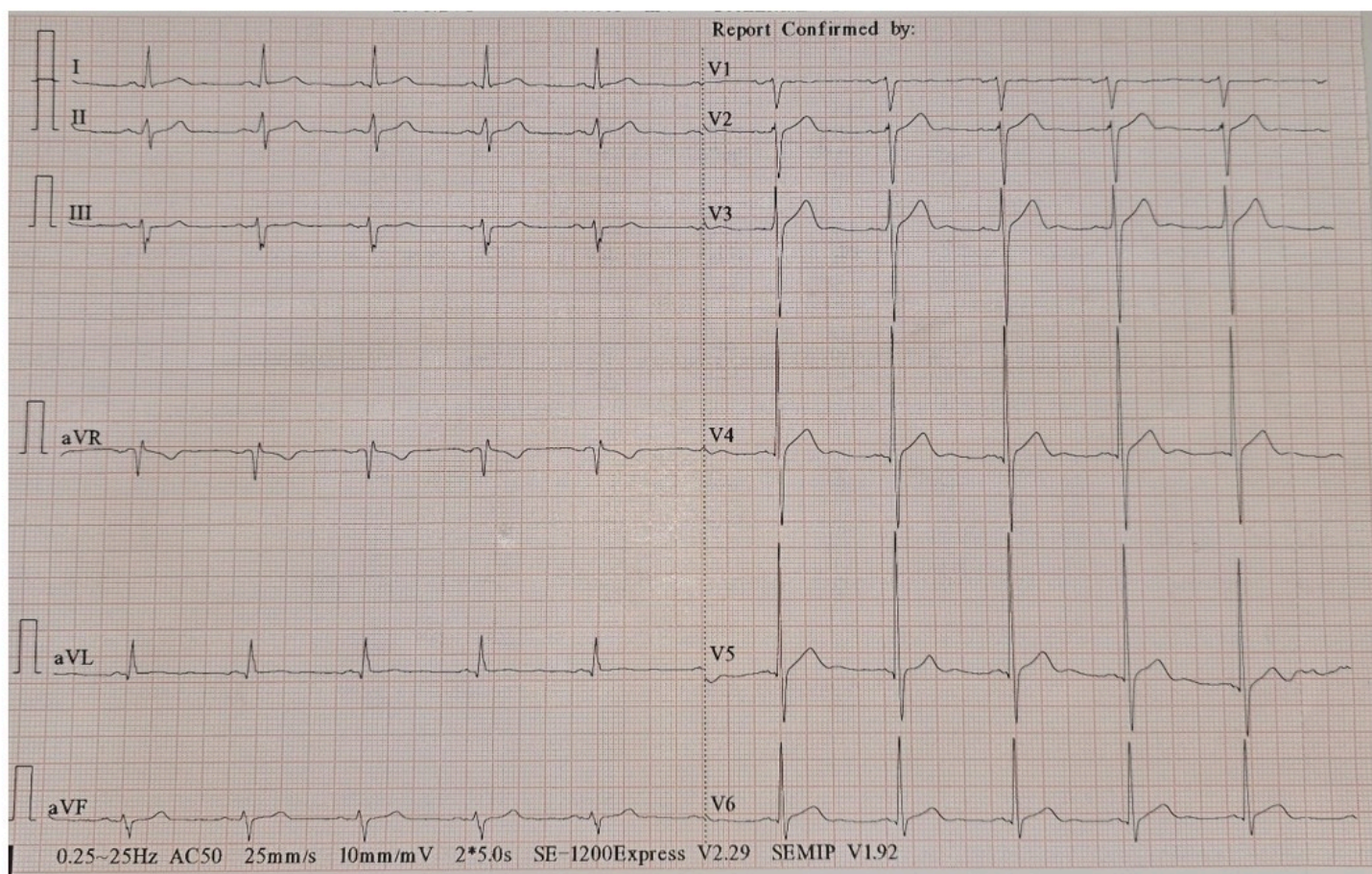


Figure 1

Twelve-lead ECG demonstrating normal sinus rhythm at 67 bpm. No ischemic changes, conduction abnormalities, or voltage criteria for left ventricular hypertrophy are present.

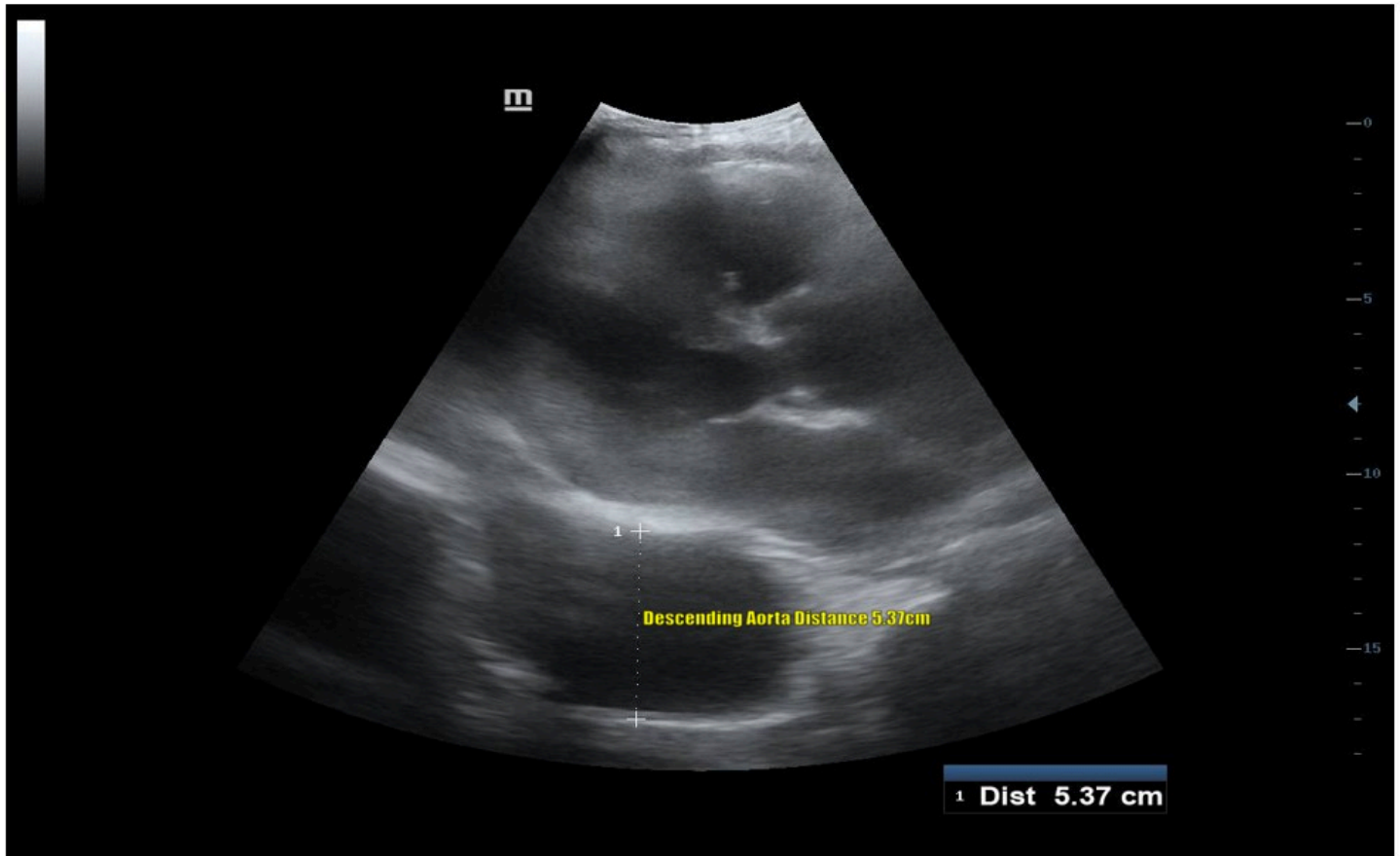


Figure 2

POCUS abdominal aorta, longitudinal view. Yellow: dilated aortic lumen (5.37 cm).

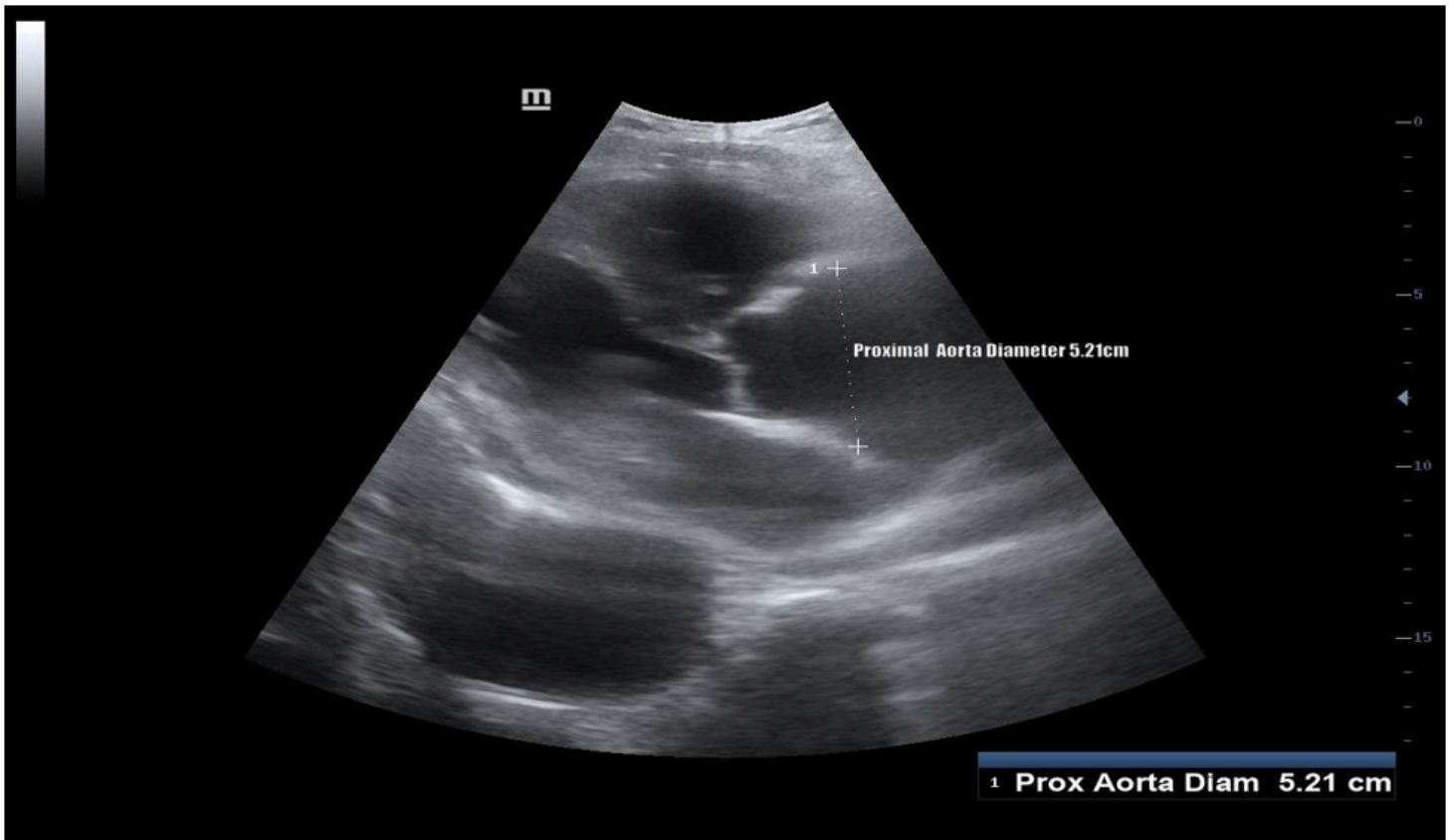


Figure 3

Proximal aortic root diameter measuring 5.21 cm on parasternal long-axis view.

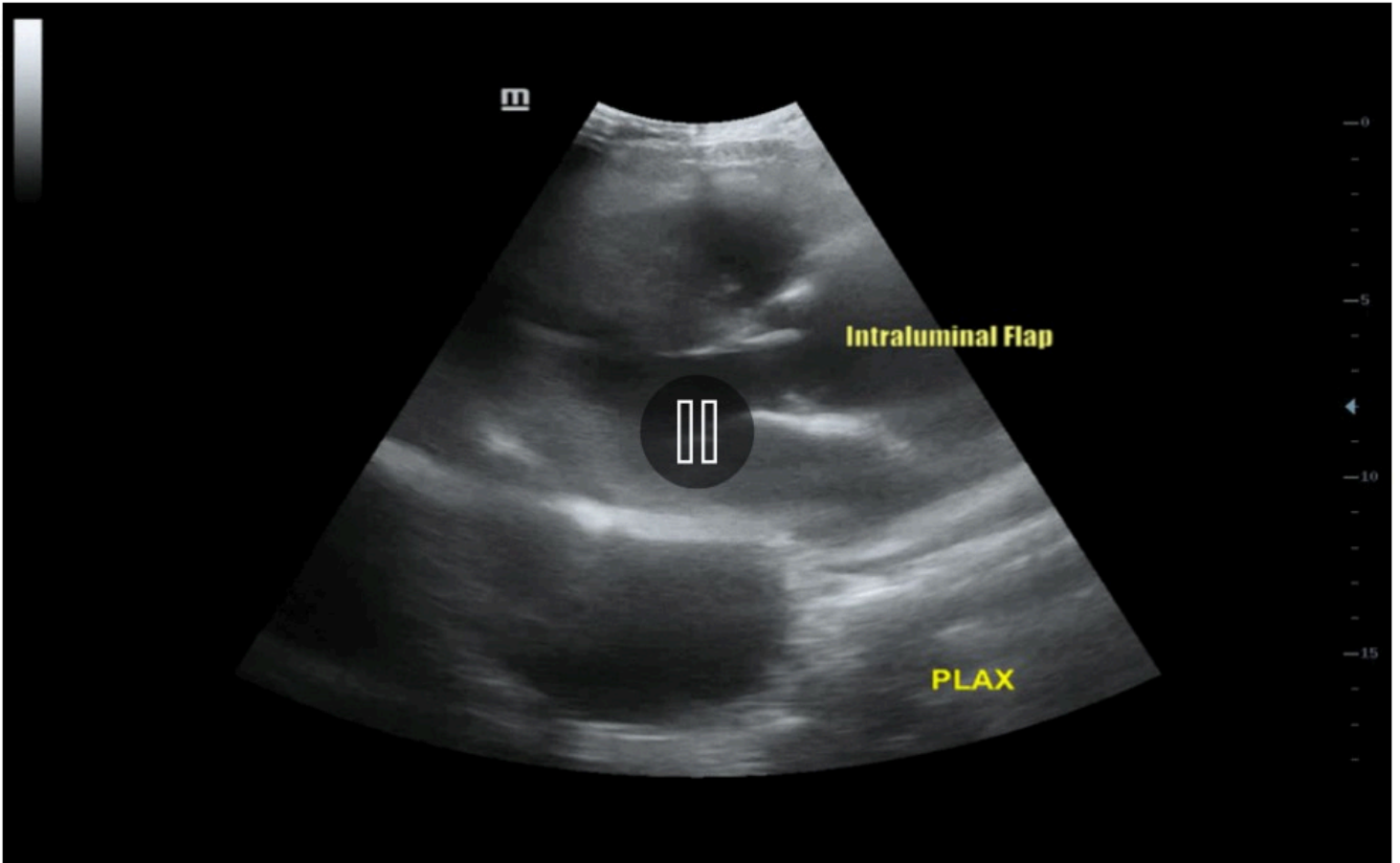


Figure 4

(Animated). PLAX view demonstrating mobile intimal flap between the aortic root and left atrium (yellow arrow).

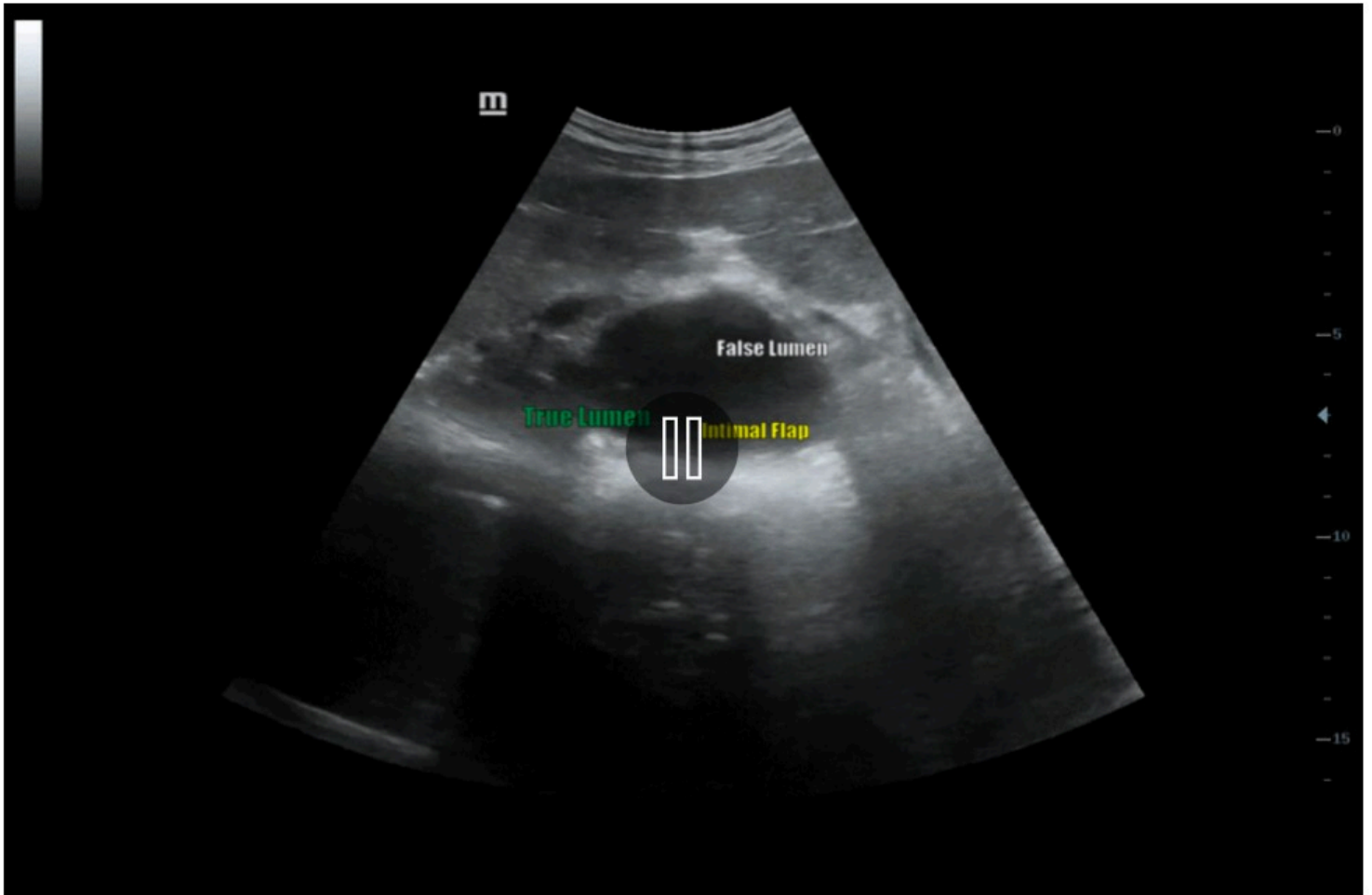


Figure 5

POCUS epigastric oblique view demonstrating true lumen (TL), false lumen (FL), and mobile intimal flap in the proximal abdominal aorta. Green overlay: true lumen (TL) – smaller, hyperechoic, with pulsatile systolic expansion. White overlay: false lumen (FL) – larger, relatively echolucent, with sluggish or absent flow. Yellow dashed line: mobile intimal flap separating TL and FL, with dynamic oscillatory flap motion visible throughout the cardiac cycle. Aortic diameter measured at 5.21 cm. These findings are highly specific for aortic dissection and constituted the key POCUS finding prompting urgent CT angiography.

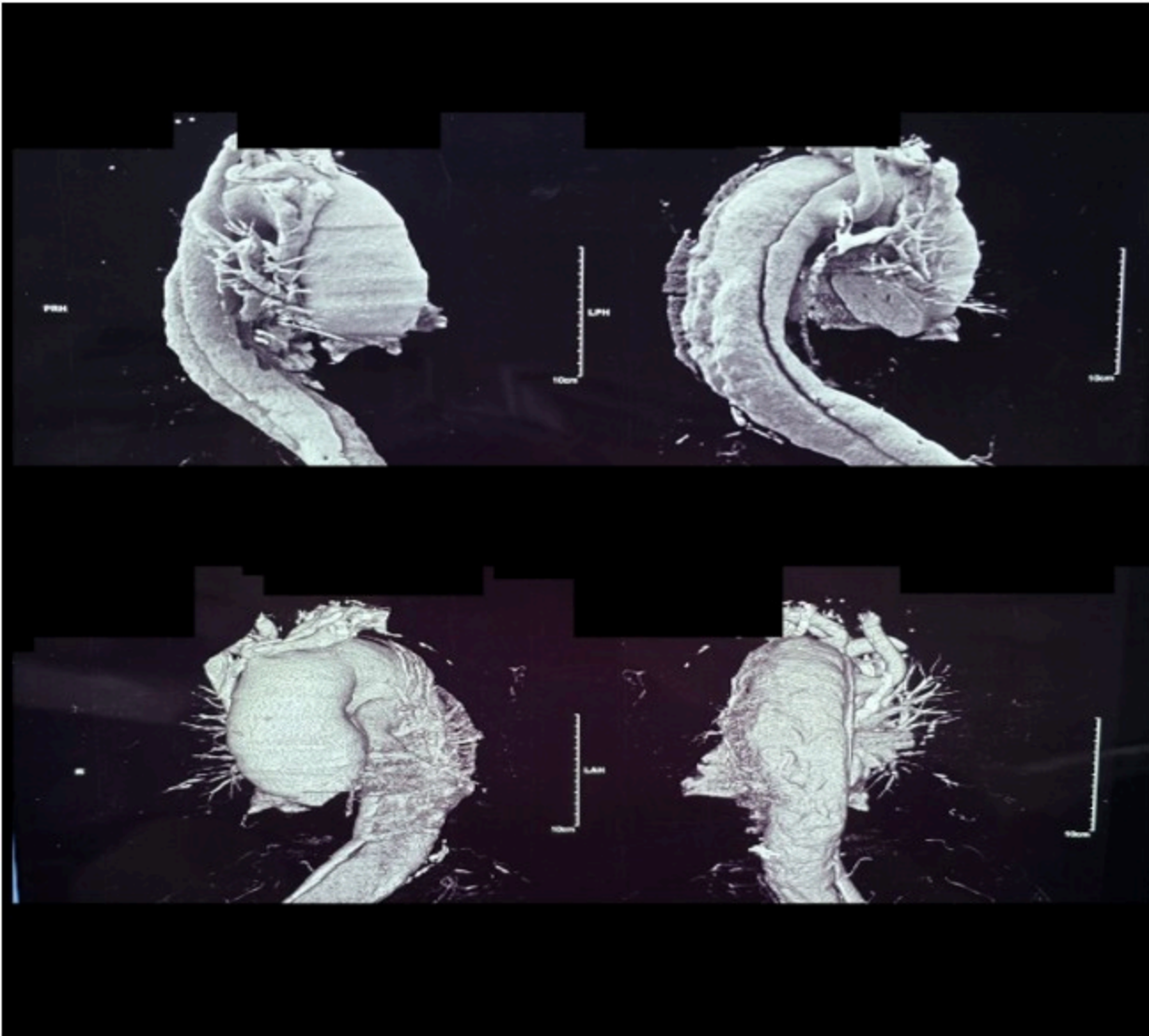


Figure 6

Three-dimensional CT angiography reconstruction demonstrating extensive Stanford type A aortic dissection with giant aneurysmal dilatation from the aortic root through the thoracoabdominal aorta. Upper panels: cardiac and arch views (PRH and LPH projections). Lower panels: thoracoabdominal extent of dissection (anterior and LAH projections).