

## Thoracic endovascular aortic repair for acute type B aortic dissection in a hypertensive woman with Conn's Syndrome: A case report

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### Abstract

**Background:** Primary aldosteronism is an increasingly recognized cause of secondary hypertension and carries disproportionately high cardiovascular risk, owing to its direct vascular and myocardial effects. Studies on aortic dissection associated with excess aldosterone remains scarce.

**Case presentation:** A 53-year-old woman with long-standing, uncontrolled hypertension presented with progressive chest and back pain. Computed tomography (CT), angiography confirmed an acute Stanford type B aortic dissection extending from the distal left subclavian artery. She was noted to have marked hypokalemia, and endocrine testing revealed primary aldosteronism due to a right adrenal adenoma. Given persistent symptoms and high-risk imaging features, thoracic endovascular aortic repair (T-EVAR) was performed. Postoperatively, she developed acute right lower limb ischemia related to vascular access injury, which required iliofemoral bypass. She recovered well with optimized antihypertensive and endocrine therapy and was discharged with plans for interval adrenalectomy.

**Conclusion:** Aldosterone-mediated vascular remodeling combined with severe hypertension may predispose patients to aortic dissection even in the absence of traditional risk factors. T-EVAR offers a safe and effective therapeutic option for appropriately selected individuals with type B dissection and concurrent endocrine hypertension.

**Keywords:** Primary aldosteronism; Conn's syndrome; Type B aortic dissection; T-EVAR; Hypertension

### 1. Introduction

Earlier termed as rare, the condition of Primary aldosteronism (PA) is now recognized as a frequently underdiagnosed unilateral or bilateral adrenal condition, characterized by overproduction of aldosterone[1,2]. PA accounts for a substantial proportion of secondary hypertension and remains subsequently prevalent among patients with resistant hypertension, with an estimated prevalence of 5- 10% among hypertensive patients [3].

In primary aldosteronism, aldosterone secretion becomes at least partly independent of its main regulator, the renin-angiotensin system, leading to suppression of circulating renin-angiotensin levels. The resulting hormone excess causes renal sodium retention, expansion of intravascular volume, and increased blood pressure, and in more severe cases hypokalemia[4]. Aldosterone excess further contributes to vascular fibrosis, endothelial dysfunction, and increased arterial stiffness, changes that can augment aortic wall stress over time [2]. Although rare, cases of aortic dissection have been documented in association with PA, suggesting aldosterone-driven hypertension may precipitate acute aortic injury in susceptible individuals[5].

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Aortic dissection is a rare but devastating vascular emergency caused by a tear in the aorta's inner lining, resulting in separation of the vessel wall layers. Acute aortic dissection (AAD) remains a life-threatening condition requiring rapid diagnosis and classification, as contemporary evidences highlight the importance of distinguishing ascending from descending aortic involvement due to major differences in prognosis and management. Current European Society of Cardiology (ESC) guidelines outline diagnostic pathways and therapeutic strategies for both uncomplicated and complicated aortic dissections [6]. Thoracic endovascular aortic repair (T-EVAR) has transformed the management of Stanford type B dissections. Randomized trails and observational studies demonstrate improved aortic remodeling and favorable long-term outcomes compared to medical therapy alone in selected patients [7-9]. Endovascular management is also particularly useful in patients with comorbid conditions such as endocrine hypertension who may be at high risk with open surgery.

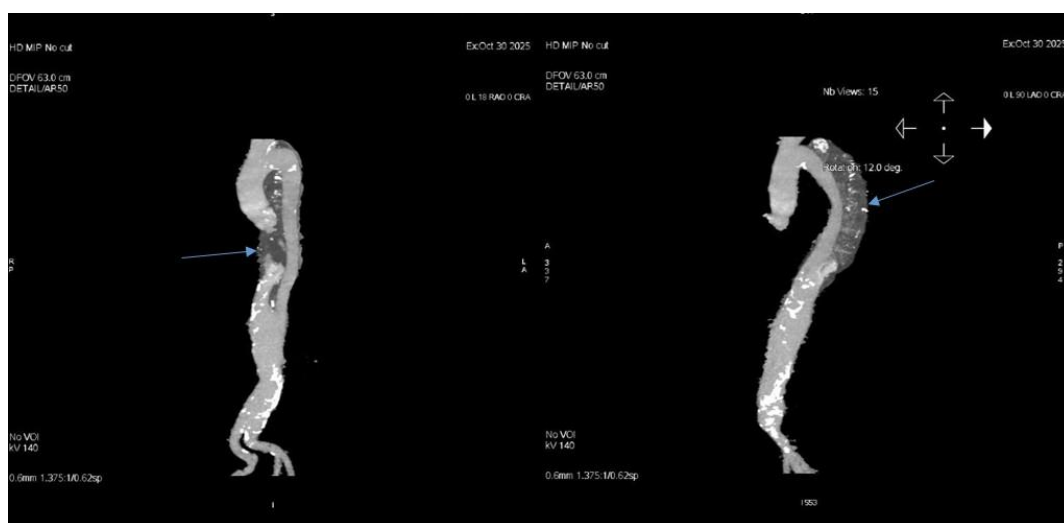
Despite growing recognition of PA, data on its association with aortic dissection and the implications for management remain limited. The existing literature on this overlap is sparse and largely confined to case reports and small series. This report hence, describes the presentation, management, and outcome of a female patient with acute type B dissection in the setting of previously undiagnosed Conn's syndrome.

## 2. Case presentation

A 53-year-old woman with a history of hypertension from the past six years, presented with recurrent, severe chest pain radiating to the back, worsening over two months. Symptoms included dyspnea on exertion, episodic blood pressure surges, palpitations, and headaches. She denied trauma, connective-tissue disorders, tobacco use, or alcohol consumption. Antihypertensive therapy prescribed elsewhere had been irregular and ineffective.

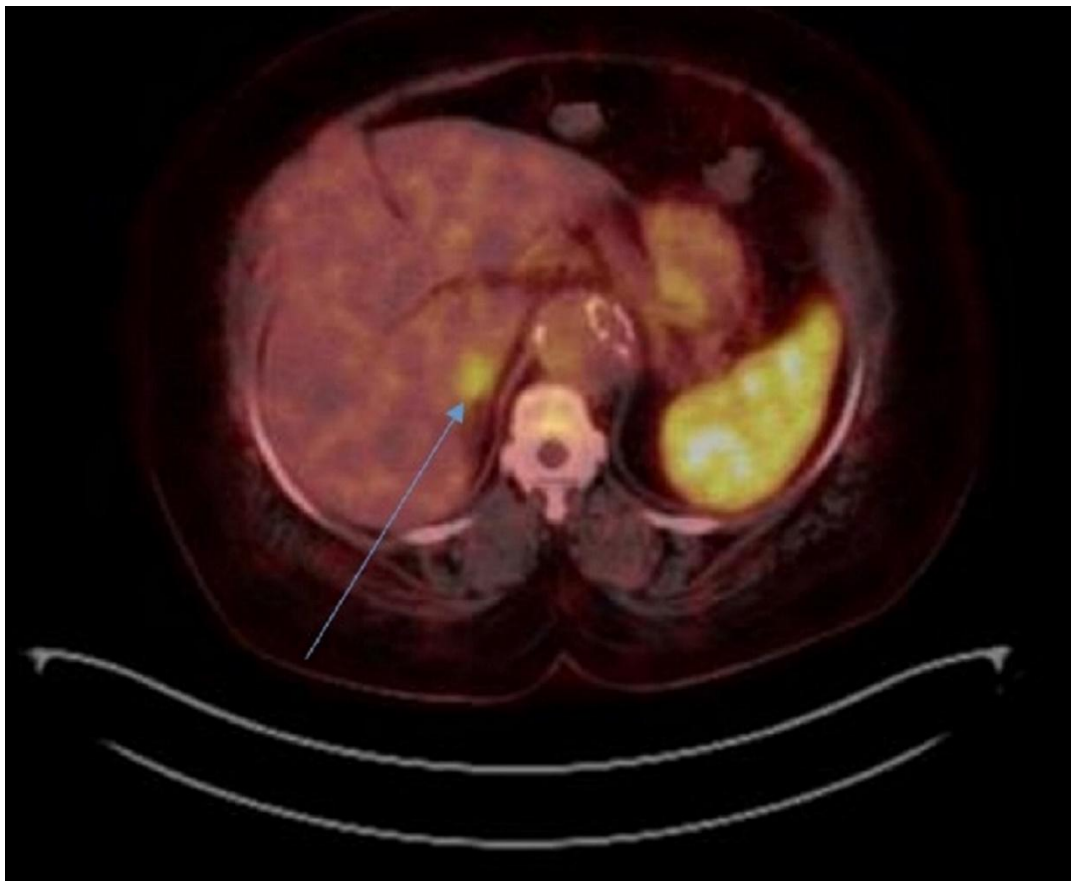
On arrival, she was markedly hypertensive but hemodynamically stable. Peripheral pulses were symmetrical, and systemic examination was unremarkable. Laboratory results demonstrated persistent hypokalemia with normal renal and hepatic function. Electrocardiograph (ECG) showed sinus rhythm; echocardiography revealed normal left ventricular function (Left Ventricular Ejection Fraction 60%) without significant valvular disease.

In view of Resistant Hypertension with hypokalemia, primary hyperaldosteronism was suspected, hence, Plasma aldosterone concentration and plasma renin activity labs were sent, which revealed Aldosterone value as 176mg/dl. Plasma Renin reports were suggestive of primary hyperaldosteronism, hence, the CT abdomen was done which revealed adenoma in the right adrenal gland measuring 2.3 x 1.6 x 2.4 cms, and a Stanford type B (DeBakey III) dissection beginning distal to the left subclavian artery, with perfusion of major visceral arteries through the true lumen. No signs of rupture or extension into the ascending aorta were present [6,10]. Figure 1 reveals CT angiogram SSD and volume rendered images reveal dissecting aneurysm on with thrombosis of the false lumen in the thoracic aorta (blue arrow).



**Figure 1** Computed tomography (CT) angiography with volume-rendered reconstructions of the thoracic aorta demonstrating a dissecting aneurysm. The true lumen is opacified, while the false lumen shows partial thrombosis (indicated by the blue arrow). The images highlight the longitudinal extent of the dissection and associated aneurysmal dilatation

A right adrenal mass prompted assessment for co-secretion of Cortisol hence was evaluated accordingly [11,12]. In view of suspected primary hyper-aldosteronism, patient was subjected to  $^{68}\text{Ga}$ -Pentixafor PET-CT. The scan identified a metabolically active adrenal nodule compatible with an aldosterone-producing adenoma confirming the diagnosis. Baseline venous duplex ultrasound of the lower limbs was normal. CT brain imaging showed only chronic small-vessel ischemic changes. Figure 2 reveals the PET-CT of the patient. PET-CT fusion images Axial and coronal reveal uptake in the right adrenal lesion. Baseline venous duplex ultrasound of the lower limbs was normal.



**Figure 2** Axial fused  $^{68}\text{Ga}$ -Pentixafor PET-CT image demonstrating focal tracer uptake in the right adrenal gland (blue arrow), corresponding to a CXCR4-expressing adrenal lesion

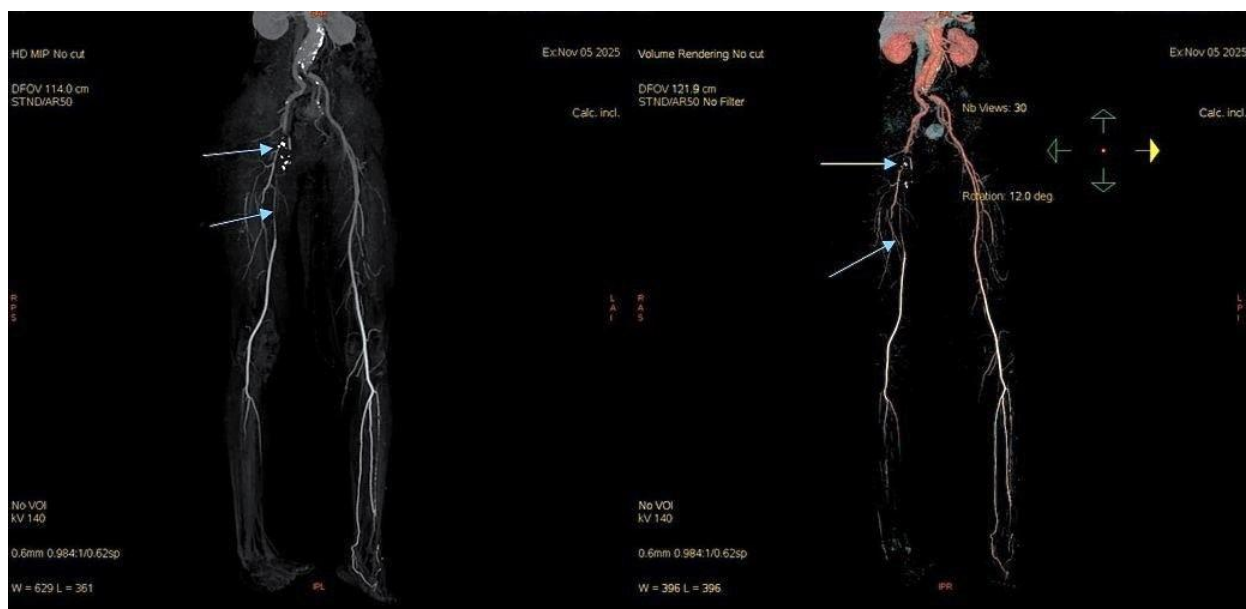
The patient was admitted to the intensive care unit for anti-impulse therapy, including intravenous beta-blockers and vasodilators, according to European Society of Cardiology (ESC) guideline recommendations for type B dissection[6]. Despite optimal therapy, she continued to experience pain and blood pressure fluctuations.

A multidisciplinary team determined that early T-EVAR due to persistent symptoms and high-risk features. Mineralocorticoid receptor antagonists and potassium supplementation were initiated for PA, with adrenalectomy planned after aortic stabilization.

On 4<sup>th</sup> November 2025, T-EVAR was performed under general anesthesia. Right femoral arterial access was obtained, and a thoracic stent graft was deployed across the primary intimal tear distal to the left subclavian artery. This approach aligns with established endovascular strategies for type B dissection[9,13].

Intraoperative angiography confirmed complete exclusion of the entry tear, restoration of true lumen flow, and absence of endo leak. Postoperative imaging demonstrated appropriate graft position and early favorable remodeling.

Within 24 hours, the patient developed acute pain and pallor in the right lower limb. Doppler ultrasound and CT angiography revealed thrombosis of the right external iliac common femoral segment, likely related to vascular access. Urgent thrombectomy using a Fogarty catheter was followed by right external iliac-to-common femoral artery bypass using an 8-mm Dacron graft. Figure 3 reveals the occlusion of the right superior femoral artery. Distal perfusion improved immediately. Recovery was uneventful, with full neurological preservation.



**Figure 3** Computed Tomography (CT) angiography with maximum intensity projection (MIP) and volume-rendered images demonstrating occlusion of the right superficial femoral artery (blue arrows), with non-opacification of the distal arterial segment and reduced distal runoff

In view of Hyperaldosteronism, patient was started on mineralocorticoids receptor antagonist, her Blood pressure and potassium levels improved significantly with mineralocorticoid receptor antagonism. [1,9] Elective adrenalectomy was planned after vascular recovery, consistent with established management of unilateral PA. [9]

The patient was discharged postoperatively in a stable condition. Antihypertensive therapy included beta-blocker, calcium-channel blocker, angiotensin converting enzyme (ACE) inhibitor, diuretic, and mineralocorticoid antagonist. Serum potassium normalized, and pulses in the right lower limb remained intact. Lifelong anti-impulse therapy, statin therapy, and dual antiplatelets were prescribed. Regular follow-up with cardiothoracic surgery and endocrinology was arranged, along with interval CT aortography.

### 3. Discussion

This case highlights the potential contribution of aldosterone excess to acute aortic pathology. Chronic hyperaldosteronism promotes arterial stiffness, collagen deposition, and endothelial dysfunction, predisposing patients to heightened aortic wall stress and, potentially, dissection [12]. Case reports have documented associations between PA and aortic dissection, supporting a pathophysiologic link between endocrine hypertension and aortic injury.

A meta-analysis comprising of 31 studies, reported that cases with primary aldosteronism reported almost three times increased risk of stroke (OR- 2.58, 95% CI- 1.93-3.45), coronary artery disease (OR-1.77, 95% CI-1.10-2.83), and atrial fibrillation (OR- 3.52, 95% CI- 2.06-5.99)[14].

Management of acute type B dissection centers on anti-impulse therapy, but persistent pain, uncontrolled hypertension, or high-risk imaging features warrant early intervention[8]. Endovascular repair has become the preferred strategy for many patients due to lower perioperative morbidity and improved aortic remodeling demonstrated in large studies and randomized trials [7,8,15,16].

Access-related complications, such as limb ischemia, although uncommon, require rapid intervention. Early recognition and timely revascularization significantly improve outcomes. Definitive management of PA with adrenalectomy is essential to prevent ongoing vascular injury. Addressing the endocrine source of hypertension reduces recurrent cardiovascular risk, including future aortic events[12]. Long-term observational data suggest that using mineralocorticoid receptor blockers or surgically treating primary aldosteronism can lower the risk of aortic complications over about five years of follow-up. Nonetheless, well-designed prospective studies are still required to prove a causal relationship and to define the best approach for screening patients with aortic dissection for primary aldosteronism.

### *List of abbreviations*

Abbreviation	Full form
AAD	Acute aortic dissection
ACE	Angiotensin converting enzyme
BP	Blood pressure
CT	Computed tomography
ECG	Electrocardiograph / Electrocardiogram
ESC	European Society of Cardiology
PA	Primary aldosteronism
PET-CT	Positron emission tomography- CT
T-EVAR	Thoracic endovascular aortic repair

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## **4. Conclusions**

Primary aldosteronism may contribute to acute aortic dissection through persistent, severe hypertension and vascular remodeling. T-EVAR provides an effective therapeutic option for anatomically suitable type B dissections, particularly in patients with complex comorbidities. A multidisciplinary approach and long-term endocrine management are critical for optimal outcomes. Early screening for PA excess in high-risk hypertensive individuals could avert progression to life-threatening vascular events, enabling timely interventions. In addition, incorporating advanced imaging and biomarker surveillance after T-EVAR can aid in assessing vascular stability and aldosterone control, reducing recurrence risks. Ultimately, tailored protocols combining vascular surgery, endocrinology, and cardiology expertise promise improved survival and quality of life in this challenging cohort.

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## **Compliance with ethical standards**

### *Disclosure of conflict of interest*

The authors declare that they have no conflicts of interest related to this case report.

### *Statement of ethical approval*

As per the policy of our institution, single-patient case reports do not require formal review by the institutional ethics committee. All procedures were conducted in accordance with institutional and international ethical standards and the Declaration of Helsinki.

### *Statement of informed consent*

The case report is fully anonymized, removing all personal information and images that could identify the patient, hence no consent is sought to publish the case report.

### *Novelty and Impact*

This case report documents the acute type B aortic dissection in a hypertensive woman with Conn's syndrome, linking aldosterone excess to vascular fragility beyond traditional risks. It highlights TEVAR's efficacy in high-risk endocrine patients, including access-site complication management and multidisciplinary care. Novel insights emphasize early PA screening in aortic emergencies, revealing aldosterone's under-recognized role in aortic wall stress and advocating tailored protocols to avert recurrence.

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### Author contributions

- Rahul Gupta- Clinical Management, Investigation, Validation, Resources, Visualization, Supervision, Writing - Review & Editing.
- Punya Pratap Kujur- Clinical Management, Investigation, Writing - Review & Editing.
- Venkataesa Reddy- Clinical Management, Investigation, Writing - Review & Editing.
- Swarup Pal- Clinical Management, Investigation, Writing - Review & Editing.
- Sneha Kothari- Clinical Management, Investigation, Writing - Review & Editing.
- Aarti Ullal- Clinical Management, Investigation, Writing - Review & Editing.
- Chintan Trivedi- Visualization, Imaging Interpretation, Writing - Review & Editing.
- Saurabh Deshpande- Visualization, Imaging Interpretation, Writing - Review & Editing.
- Umerahmed Khan - Resources, Data Curation, Writing - Original Draft, Project Administration, Ethics & Consent, Writing - Review & Editing.
- Alisha Shah- Writing - Review & Editing

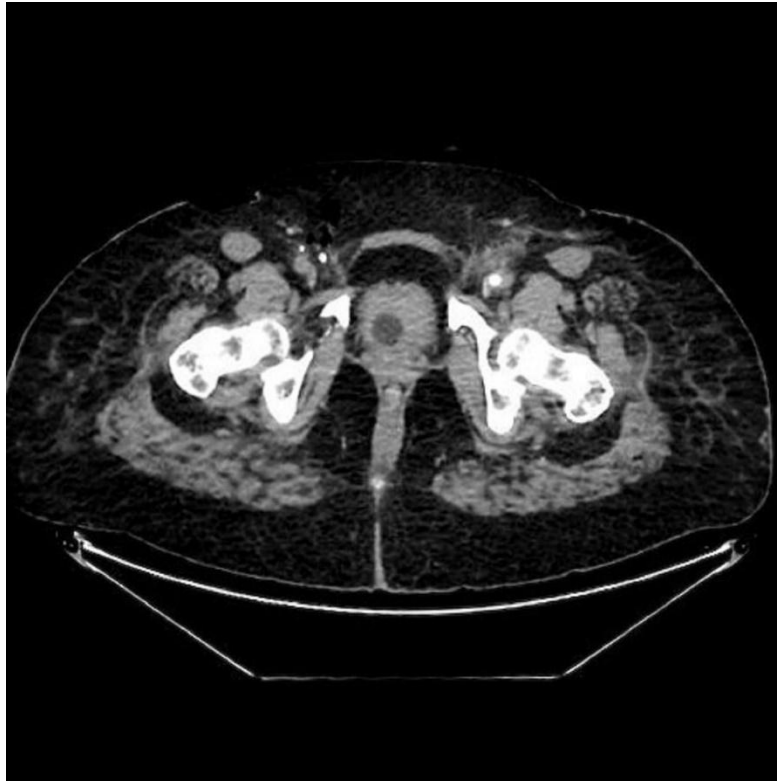
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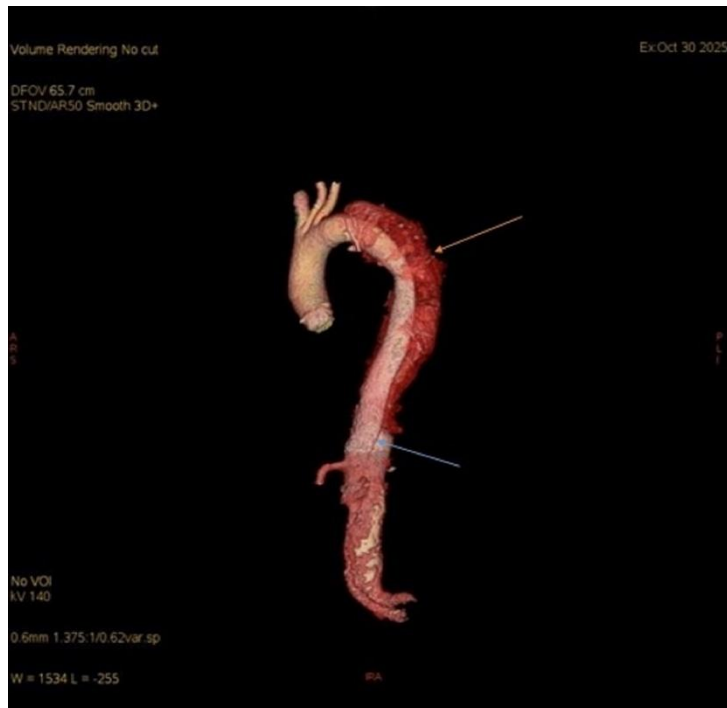
## Appendix



**Figure 4** Axial contrast-enhanced CT image demonstrating a filling defect within the right superficial femoral artery, consistent with intraluminal thrombus



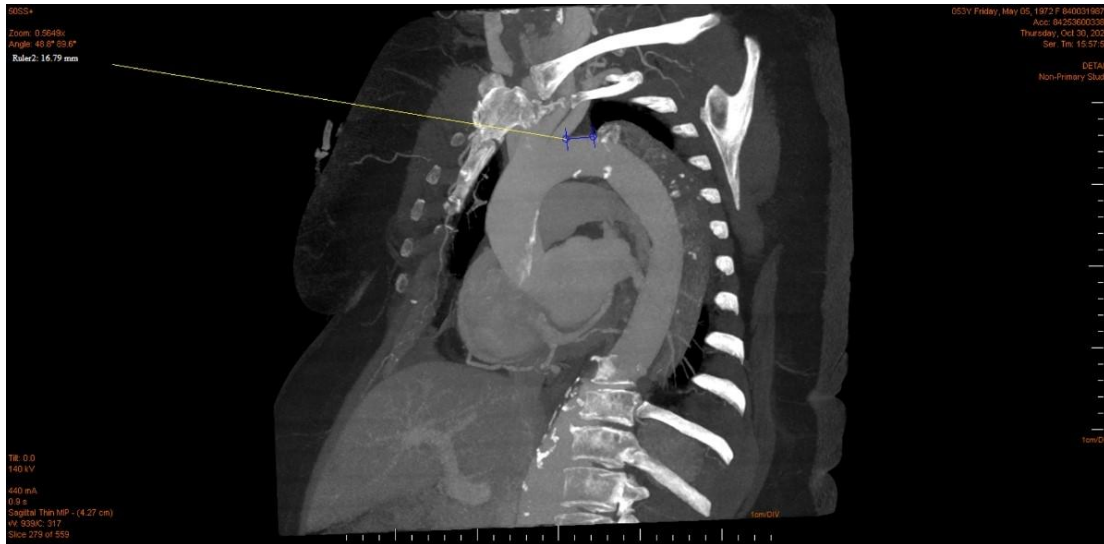
**Figure 5** Axial contrast-enhanced CT image demonstrating a dissecting aneurysm of the thoracic aorta with a partially thrombosed false lumen (orange arrow) and an opacified true lumen (blue arrow)



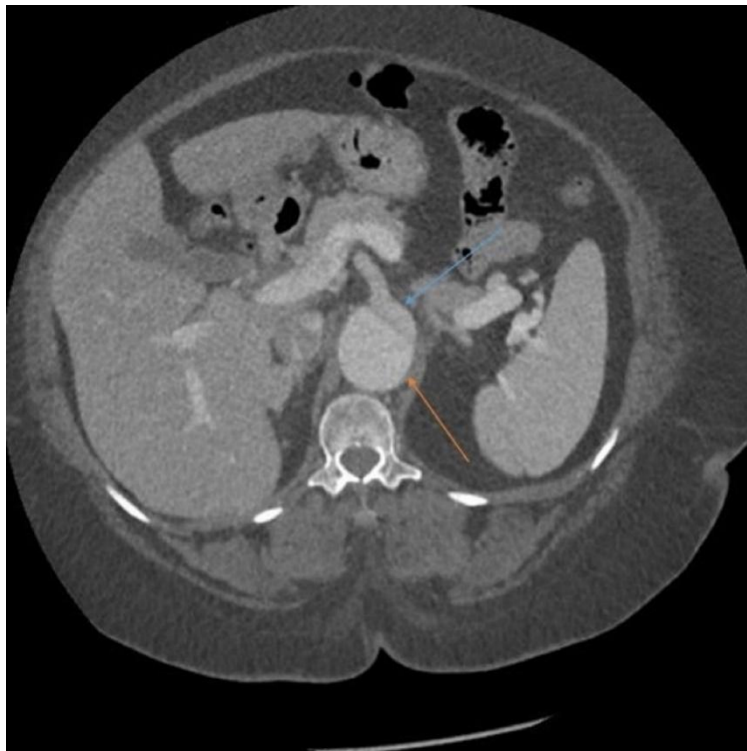
**Figure 6** Volume-rendered CT angiography image of the thoracic aorta demonstrating a dissecting aneurysm with a partially thrombosed false lumen (orange arrow) and a contrast-opacified true lumen (blue arrow), delineating the extent of the dissection



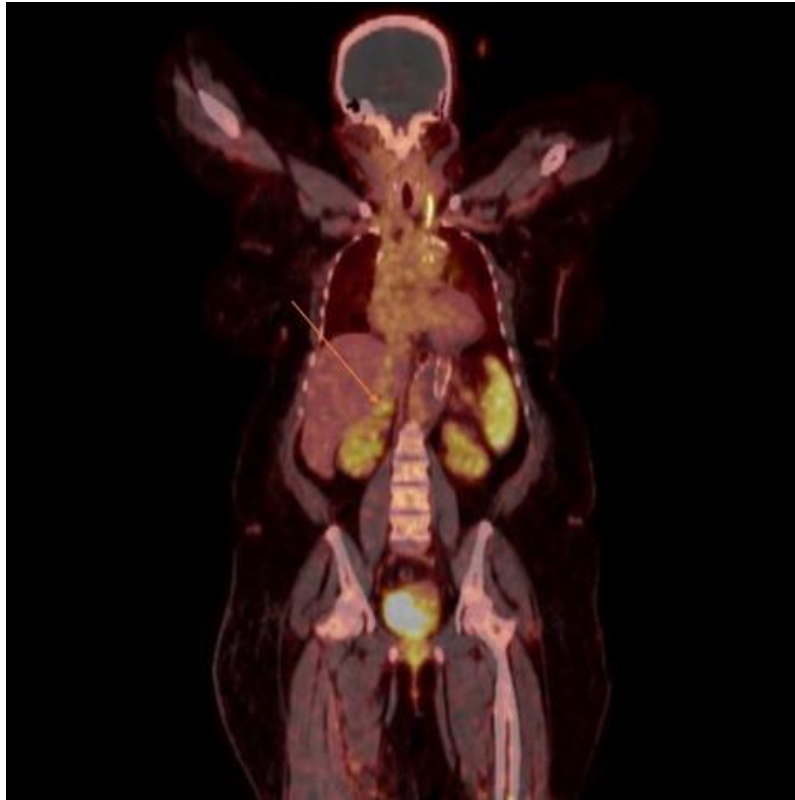
**Figure 7** Axial contrast-enhanced CT image demonstrating a nodular enhancing lesion within the right adrenal gland (arrow), suggestive of an adrenal mass lesion



**Figure 8** Curved multiplanar reformatted CT angiography image demonstrating the distance between the origin of the left subclavian artery and the proximal extent of the aortic dissection (measurement line), aiding in procedural planning and landing zone assessment



**Figure 9** Axial contrast-enhanced CT image demonstrating an aortic dissection flap extending into the abdominal aorta, with the coeliac artery arising from the true lumen (blue arrow) and the false lumen identified adjacent to it (orange arrow)



**Figure 10** Coronal fused Positron Emission Tomography Computed Tomography (PET-CT) image demonstrating increased fluorodeoxyglucose (FDG) uptake in the right adrenal gland (arrow), consistent with a metabolically active adrenal lesion