Staged thoracic endovascular aortic repair for an extensive Stanford type B aortic dissection in a high-risk patient with chronic kidney disease. A case report

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Case Report

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Background: Objective: To describe the endovascular management of an extensive Stanford type B aortic dissection (TBAD) in a high-risk patient, treated with thoracic endovascular aortic repair (TEVAR) as the first stage of a staged repair.
Introduction: TBAD is a life-threatening acute aortic syndrome. TEVAR is recommended for complicated disease and should be considered in uncomplicated cases with high-risk features.
Case presentation: A 66-year-old man with hypertension, type 2 diabetes mellitus, ischemic heart disease and stage 3a chronic kidney disease presented with epigastric pain, nausea and vomiting. Computed tomography angiography showed a TBAD starting distal to the left subclavian artery, with a maximum diameter of 49 mm and a false lumen extending to the iliac arteries. Elective TEVAR was performed five months after symptom onset through a percutaneous femoral access preclosed with Perclose ProStyle. Intravascular ultrasound (IVUS) was used to differentiate the true and false lumens, and two thoracic stent grafts (42 × 42 × 150 mm and 44 × 40 × 150 mm) were deployed from the aortic arch at the origin of the left subclavian artery to proximal to the celiac trunk. He was discharged home improved, with a second stage planned for the residual distal dissection.
Discussion: IVUS guidance and percutaneous access allowed a safe proximal repair in a patient with renal impairment; the residual thoracoabdominal false lumen requires staged treatment and close imaging surveillance.
Conclusion: Staged TEVAR is a feasible option for extensive TBAD in high-risk patients.

Keywords: Aortic Dissection; Endovascular Procedures; Blood Vessel Prosthesis.

Acute aortic syndrome comprises a group of life-threatening aortic pathologies in which aortic dissection is the most frequent entity; its management requires prompt diagnosis and multidisciplinary care, ideally in high-volume aortic centers (1). The incidence of aortic dissection is estimated at 5 to 30 cases per million people per year, and classic acute aortic dissection has been reported at 2.6–3.5 cases per 100,000 person-years (2). Stanford type B aortic dissection (TBAD) involves the descending aorta distal to the left subclavian artery and, according to the time elapsed from symptom onset, is classified as hyperacute (<24 h), acute (1–14 days), subacute (15–90 days) or chronic (>90 days) (3).

Current guidelines recommend thoracic endovascular aortic repair (TEVAR) for complicated TBAD (malperfusion, rupture, rapid expansion, or refractory pain or hypertension) and consider it reasonable in uncomplicated TBAD with high-risk features, such as a maximal aortic diameter >40 mm, a false-lumen diameter >20–22 mm or an entry tear >10 mm (3). The recently updated European vascular surgery guidelines also address the management of descending thoracic and thoracoabdominal aortic disease (4). In uncomplicated TBAD, TEVAR improves aortic remodeling, although definitive evidence of a survival benefit over optimal medical therapy is still lacking (5), and the timing of intervention appears to influence long-term outcomes (6).

Dissections that extend from the descending thoracic aorta to the iliac bifurcation pose an additional challenge, because coverage of the entire dissected segment in a single procedure increases its complexity, and the visceral arteries may arise from the false lumen. We present the case of a high-risk patient with chronic kidney disease and an extensive TBAD treated with IVUS-guided percutaneous TEVAR as the first stage of a staged repair. This report was prepared following the CARE guidelines.

Case report

A 66-year-old retired man residing in Tijuana, Baja California, was evaluated at the Hospital General ISSSTE "Fray Junipero Serra". His medical history included systemic arterial hypertension diagnosed in 2020, treated with losartan 50 mg once daily, metoprolol 100 mg once daily, nifedipine 60 mg every 12 h and isosorbide dinitrate 10 mg every 8 h; dual antiplatelet therapy with clopidogrel 75 mg and acetylsalicylic acid 100 mg daily; type 2 diabetes mellitus diagnosed in 2020, currently without pharmacological treatment; and ischemic/hypertensive heart disease with left ventricular hypertrophy and a left ventricular ejection fraction (LVEF) of 55%. He also had chronic kidney disease (CKD) without renal replacement therapy, stage 3a with an estimated glomerular filtration rate (CKD-EPI) of 59 mL/min/1.73 m² in April 2026; a renal ultrasound (November 2025) showed a small right kidney with

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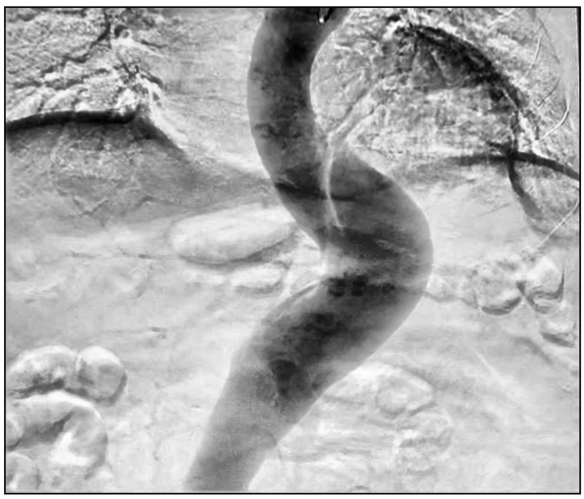


Figure 1. Intraoperative digital subtraction aortography of the descending thoracic aorta (August 28, 2026), showing the dilated and tortuous dissected segment. Aortography documented the dissection beginning distal to the origin of the left subclavian artery, with multiple re-entry tears. Patient identifiers were removed from the images.

loss of corticomedullary differentiation and a normal left kidney. Other history included untreated dyslipidemia, active smoking (14 cigarettes per day since the age of 25 years), marijuana use in his youth, open reduction and internal fixation of an ankle fracture, and allergy to penicillin.

At the end of March 2026, he presented to the emergency department with epigastric pain, nausea and vomiting, and elevated blood pressure was documented. On April 16, 2026, abdominal ultrasound showed chronic calculous cholecystitis, grade II hepatic steatosis and an increased diameter and tortuosity of the abdominal aorta, so computed tomography angiography (CTA) of the aorta was performed the same day. CTA showed an ascending aorta of preserved caliber (38 mm) that increased to 49 mm distal to the origin of the supra-aortic trunks, with an intraluminal intimal flap creating a false lumen that extended to the internal iliac arteries; the celiac trunk, the right renal artery and the inferior mesenteric artery arose from the false lumen. Additional findings were a type II hiatal hernia, colonic diverticulosis and loss of height of the L1 vertebral body. A diagnosis of Stanford TBAD was established.

The patient was admitted for strict blood pressure control and evaluated by the Angiology service, who indicated endovascular repair. The cardiology preoperative evaluation (April 21, 2026) reported Goldman class II, Detsky class II and an American Society of Anesthesiologists (ASA) class 2-3, and recommended a perioperative systolic blood pressure of 100–120 mmHg. After hemodynamic stabilization, he was discharged on medical therapy. The procedure was subsequently scheduled electively, and on August 25, 2026, he was readmitted.

On readmission he was asymptomatic, with a heart rate of 78 bpm, respiratory rate of 18 rpm, blood pressure of 133/78 mmHg and temperature of 36.5 °C. He was alert and oriented, with normal cardiopulmonary examination, a soft, non-tender abdomen and palpable, symmetrical peripheral pulses without signs of distal ischemia. Weight was 80 kg and height 1.67 m (calculated body mass index 28.7 kg/m²). Preoperative laboratory tests showed hemoglobin 13.7 g/dL, platelets 175 × 10³/µL, international normalized ratio (INR) 1.01, glucose 126 mg/dL, urea 48.7 mg/dL and creatinine 2.05 mg/dL. The electrocardiogram showed sinus rhythm with left anterior fascicular block, and the chest radiograph showed a prominent aortic knob with mediastinal

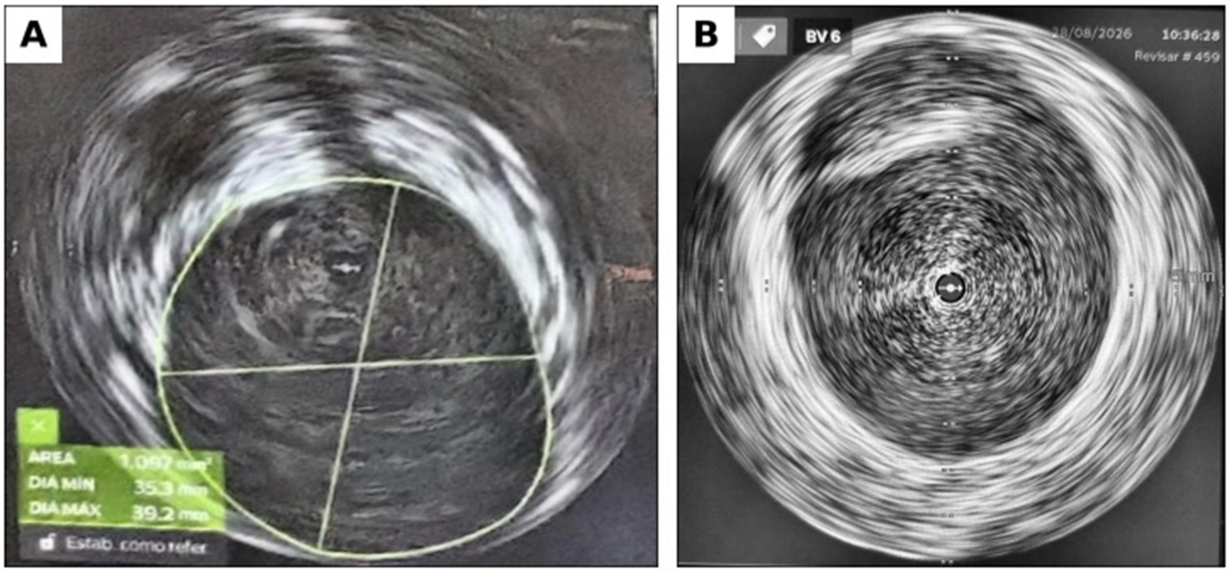


Figure 2. Intraoperative intravascular ultrasound (IVUS) of the descending thoracic aorta (August 28, 2026), used to differentiate the true lumen from the false lumen. (A) Cross-sectional image with a luminal area of 1,097 mm² and minimum and maximum diameters of 35.3 mm and 39.2 mm. (B) Cross-sectional IVUS image of the descending thoracic aorta with the 5-mm calibration scale. Patient identifiers were removed from the images.

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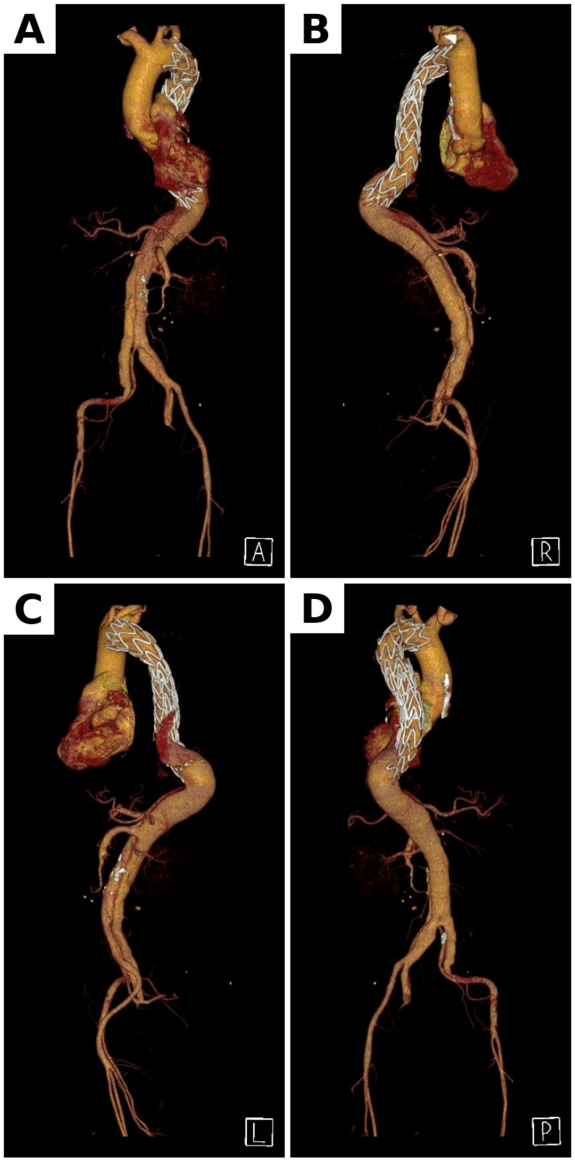


Figure 3. Postoperative computed tomography angiography, three-dimensional volume-rendered reconstructions (September 1, 2026). (A) Anterior, (B) right, (C) left and (D) posterior views showing the two thoracic stent grafts deployed from the aortic arch at the origin of the left subclavian artery to proximal to the celiac trunk; the abdominal aorta, its visceral branches and the iliac arteries are also displayed.

widening. Internal medicine (August 27, 2026) reported Goldman II, Detsky II and a Caprini score of 4; anesthesiology classified the patient as ASA III; and cardiology (August 28, 2026) classified the procedure as high-risk surgery, with an AUB-HAS2 score of 4 points and an estimated 17.5% risk of a postoperative adverse event.

Surgical technique

On August 28, 2026, approximately five months after symptom onset (chronic phase), the patient underwent elective TEVAR in the operating room under general anesthesia, performed by the Angiology service with the participation of the General Surgery residents. After asepsis and antisepsis of the right inguinal and left radial regions and placement of sterile drapes, the right common femoral artery and the left radial artery were punctured under fluoroscopic guidance. An 8 Fr sheath was placed in the femoral artery after preclosure with two Perclose ProStyle suture-mediated closure devices (7), and a 6 Fr sheath was placed in the left radial artery.

Aortography with a 6 Fr pigtail catheter documented the dissection beginning distal to the origin of the left subclavian artery, with multiple re-entry tears extending distally to the iliac bifurcation (Figure 1). Intravascular ultrasound (IVUS) of the descending thoracic aorta was used to differentiate the true lumen from the false lumen, documenting a luminal area of 1,097 mm² with minimum and maximum diameters of 35.3 mm and 39.2 mm, respectively (Figure 2A, 2B) (8).

A high-support guidewire was advanced to the proximity of the aortic valve and the femoral access was upsized to a 26 Fr sheath. A first thoracic stent graft of 42 × 42 × 150 mm was advanced and deployed, followed by a second thoracic stent graft of 44 × 40 × 150 mm deployed distally. The proximal stent graft was landed in the aortic arch at the level of the origin of the left subclavian artery, and the distal stent graft ended proximal to the origin of the celiac trunk. Completion aortography showed adequate position of both stent grafts, with preservation of flow through the left subclavian artery and the celiac trunk. The 26 Fr sheath was removed and the arteriotomy was closed with the previously placed ProStyle sutures; hemostasis of the access site was verified and the skin was closed with Sarnoff sutures of 2-0 nylon. Estimated blood loss was 100 mL, no drains were placed and the textile count was complete. The procedure was completed without intraoperative complications and the patient was transferred in stable condition to the intensive care unit (ICU).

Because the dissection extended to the iliac bifurcation, a staged strategy was planned: this first stage treated the thoracic segment, from the aortic arch at the origin of the left subclavian artery to proximal to the celiac trunk, and a second stage was planned for the residual thoracoabdominal and abdominal dissection.

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. The patient's identity was protected; all personal identifiers were removed from the text and images.

On arrival at the ICU, the patient was awake and neurologically intact, reporting mild pain at the left radial puncture site. Heart rate was 51 bpm and blood pressure 170/90 mmHg, so an intravenous

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nitroglycerin infusion was started, titrated up to 20 µg/min, and losartan was added to nifedipine, with targets of systolic blood pressure <120 mmHg and heart rate of 60–80 bpm; beta-blockers were temporarily withheld because of sinus bradycardia. Point-of-care cardiac ultrasound showed preserved global contractility without pericardial effusion. Oxygen saturation was 97% with nasal cannula at 2 L/min, and the portable chest radiograph showed the thoracic stent graft in adequate position without pneumothorax or significant pleural effusion. Urine output was 1.5 mL/kg/h. Antibiotic prophylaxis with ceftriaxone was administered. The right inguinal access was managed with compression and a counterweight for 6 h; an area of induration was marked for surveillance as a probable hematoma.

On postoperative day 1, laboratory tests showed leukocytes 12.9 × 10^3/µL with 85.5% neutrophils, hemoglobin 14.0 g/dL and platelets 161 × 10^3/µL, without clinical evidence of infection. Serum creatinine remained stable compared with the preoperative value (2.12 and 2.00 mg/dL on postoperative day 1 vs 2.05 mg/dL preoperatively), and creatine kinase was 40.1 U/L with a CK-MB of 18 U/L. He was transferred to the surgical ward the same day.

On postoperative day 3, he was asymptomatic, tolerating an oral diet, with a heart rate of 79 bpm, blood pressure of 105/63 mmHg, oxygen saturation of 98% and temperature of 36.7 °C. The right inguinal puncture site showed ecchymosis without progression and without evidence of hematoma expansion, and the dorsalis pedis and posterior tibial pulses were present bilaterally. A control CTA was performed on September 1, 2026; the three-dimensional volume-rendered reconstructions show both stent grafts deployed in the descending thoracic aorta, from the aortic arch to the level of the celiac trunk, with contrast opacification of the abdominal aorta, its visceral branches and the iliac arteries (Figure 3A–3D). This study was used to confirm the position of the stent grafts before discharge.

On September 2, 2026 (postoperative day 5; hospital stay 8 days), the patient was discharged home in improved condition, hemodynamically stable, without supplemental oxygen, without pain, with the surgical wound free of infection and without nosocomial infection. Outpatient follow-up was scheduled with Angiology (September 2026), Cardiology and Nephrology (November 2026). At the time of writing, the patient remains under outpatient follow-up for scheduling of the second surgical stage.

Discussion

The present case illustrates the endovascular management of an extensive TBAD in a patient with multiple cardiovascular, metabolic and renal comorbidities, in whom a staged strategy was chosen. The initial CTA showed a maximal aortic diameter of 49 mm, a high-risk imaging feature for which current guidelines consider TEVAR reasonable (2), in a patient who also had visceral arteries arising from the false lumen and a dissection extending to the iliac bifurcation.

The timing of TEVAR is a relevant point. In a meta-analysis of reconstructed time-to-event data including 4,793 patients, Sá et al. found timing-specific differences in outcomes, pointing to the subacute phase as the optimal moment to achieve better long-term results (6). In our patient, the indication was established during the subacute phase, and the procedure was performed electively in the chronic phase, after a period of hemodynamic stabilization under medical therapy. Medical therapy aimed at the targets recommended by the guidelines, systolic blood pressure <120 mmHg and heart rate of 60–80 bpm (2) or ≤60 bpm (3), remains the cornerstone of management. In uncomplicated TBAD, a meta-analysis of 25,650 patients showed improved aortic remodeling after TEVAR, although without definitive evidence of increased survival compared with optimal medical therapy alone (5).

Another technical aspect worth mentioning is the use of intravascular ultrasound (IVUS) to identify the true and false lumens in the descending aorta, a critical step in aortic dissection to ensure that the guidewire and the stent graft are delivered within the true lumen. In a meta-analysis of 4,219 patients undergoing TEVAR or endovascular aneurysm repair, IVUS was associated with a reduction in contrast volume (34.65 mL) and fluoroscopy time (6.13 min), with similar rates of endoleak, reintervention and mortality (8). This potential reduction in contrast exposure is particularly relevant in a patient with CKD.

Renal function was a major concern. In the International Registry of Acute Aortic Dissection, acute kidney injury (AKI) occurred in 16.2% of patients after TEVAR for acute TBAD, was associated with higher in-hospital morbidity and mortality, and a history of CKD was independently associated with postoperative AKI (9). In our patient, with a baseline creatinine of 2.05 mg/dL, serum creatinine remained stable during the early postoperative period (2.12 and 2.00 mg/dL), and urine output was preserved.

Finally, the first stage sealed the thoracic segment from the aortic arch at the origin of the left subclavian artery to proximal to the celiac trunk, while the dissection persists distally to the iliac bifurcation. TEVAR aims to seal the proximal entry tear and promote false-lumen thrombosis; however, satisfactory false-lumen thrombosis and remodeling may be achieved in as few as 40% of patients, and

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remodeling is poorer in the abdominal aorta, where persistent false-lumen perfusion increases the risk of aneurysmal degeneration (10). For this reason, extension of the repair distally is often required, and several endovascular techniques have been described to manage a patent distal false lumen (10). Imaging surveillance is mandatory after TEVAR: CT at 1 and 12 months and annually thereafter (2), or at 1, 6, 12, 24, 36, 48 and 60 months according to European recommendations (3). In our patient, the visceral arteries arising from the false lumen make the planning of the second stage particularly important.

Conclusion

IVUS-guided percutaneous TEVAR was a safe first stage in the management of an extensive Stanford type B aortic dissection in a high-risk patient with chronic kidney disease, without intraoperative complications, without early deterioration of renal function and with discharge on postoperative day 5. In dissections extending to the iliac bifurcation, a staged approach allows the thoracic segment to be treated first, while the residual distal dissection requires planned completion and strict imaging surveillance.

Conflicts of interests

None declared by the authors.

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