CASE REPORT

Double acute aortic syndrome in a patient with end-stage chronic kidney disease - Case report and literature review

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Introduction: Cardiovascular diseases are the leading cause of mortality worldwide, with aortic dissection being one of the most serious conditions, associated with high mortality. According to the Stanford classification, dissections are grouped into type A, which involves the ascending aorta and requires immediate surgery, and type B, located distal to the left subclavian artery, for which treatment may be both medical and/or endovascular. End-stage chronic kidney disease, treated by hemodialysis, is a severe risk factor owing to treatment-resistant hypertension, vascular calcifications, and systemic fragility.

Case presentation: We present the case of a 44-year-old patient with end-stage chronic kidney disease, dependent on hemodialysis for 14 years and with hypertension refractory to treatment, with a literature review. At the first admission, the initial CT angiography revealed an extensive Stanford type B dissection with severe vascular damage for which a hybrid procedure was performed: debranching of the supra-aortic vessels, followed by thoracic endovascular repair. Two months postoperatively, due to difficult-to-control hypertension, she was readmitted for a Stanford type A dissection, requiring a redo sternotomy, replacement of the ascending aorta with a Dacron graft and re-vascularization of the supra-aortic branches. Despite severe comorbidities and dialysis dependence, the patient survived both interventions and was discharged in stable condition.

Conclusions: Acute aortic dissection in dialysis-dependent patients is rare but carries exceptionally high mortality. The coexistence of end-stage renal disease, refractory hypertension, and vascular calcification increases diagnostic and therapeutic challenges. Hybrid or emergency surgical–endovascular approaches can be life-saving and multidisciplinary management with careful long-term follow-up are essential to optimize prognosis.

Keywords: acute aortic dissection, end-stage chronic kidney disease, hybrid surgical-endovascular management

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Introduction

Acute aortic syndromes are an extremely serious cardiovascular conditions with multifactorial etiology, including genetic, degenerative, atherosclerotic and inflammatory factors and, despite advances in diagnostic imaging, it is still a life-threatening condition, with an estimated incidence of 3.5-14 per 100,000 person-years and a hospital mortality rate of about 27% [1].

Together with intramural hematoma and penetrating aortic ulcer, aortic dissection (AD) belongs to the spectrum of acute aortic syndromes (AAS), which are associated with high early mortality, with the prognosis of these patients dramatically decreasing, reaching a mortality estimated at 1-2% per hour if left untreated in type A dissections [2].

Beyond complex clinical and surgical management, AAS also poses a significant economic challenge, with a major economic impact on the healthcare system through the high costs of diagnosis, treatment and long-term post-operative care, with end-stage renal disease (ESRD) further increasing cardiovascular morbidity and mortality, as well as healthcare resource utilization [3,4].

Hypertension is both a leading cause and a consequence for chronic kidney disease (CKD), with more than 70% of AD patients having a history of high blood pressure and refractory to drug treatment, making this group particularly vulnerable to dissection and complicating surgical management [5,6].

However, emergency surgical repair in dialysis patients with acute type A aortic dissection remains feasible and may save lives, despite higher perioperative mortality and long-term risks compared to non-ESRD patients [7].

Case presentation

We present the case of a 44-year-old woman with a complex medical history, sudden-onset abdominal pain and critically high blood pressure (230/100 mmHg). She had no prior history of aortic endovascular intervention that could favor the acute aortic syndrome. Upon admission, the patient was diagnosed with acute aortic dissection type Stanford B, grade III treatment-resistant secondary hypertension, hypertensive cardiomyopathy, hypertensive

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microangiopathy, mild-to-moderate degenerative mitral regurgitation, history of hypertensive pulmonary edema, chronic kidney disease requiring dialysis, electrolyte imbalances, depression under treatment, and hypercholesterolemia.

Clinical examination revealed bilaterally diminished peripheral pulses without peripheral edema, intact precordial skin and regular heart sounds. Extremely high blood pressure required immediate initiation of intravenous antihypertensive therapy. Laboratory data showed total protein of 6.1 g/dL, CRP 6.07 mg/dL, creatinine 7.94 mg/dL, urea 79,18 mg/dL, LDH 270 U/L, and oxygen saturation 89%. Preoperative ECG showed sinus rhythm, left axis deviation, and signs of left ventricular hypertrophy with a strain pattern. Preoperative echocardiography did not reveal dissection of the ascending aorta or aortic arch. Measurements included an ascending aorta diameter of 39 mm, interventricular septum of 19 mm, diameter of the left ventricle of 47 mm, ejection fraction of 55 mm, right atrium of 62 mm, and left atrium of 70 mm. Moderate mitral regurgitation with aortic peak velocity 2.2 m/s was present. The inferior vena cava measured 28 mm without respiratory collapse. The abdominal aorta showed calcified atherosclerotic changes at the origin of the superior mesenteric artery (SMA), with suspicion of distal dissection and no pericardial effusion.

The reference imaging – preoperative thoraco-abdominal CT angiography revealed subtle motion artifacts in the ascending aorta, without signs of aneurysm or dissection. Starting at the aortic arch near the origin of the left subclavian artery, a dissecting flap extended across the entire descending thoracic and abdominal aorta, showing a 180-degree twist. The dissection involved the origin of the celiac trunk, with a mural thrombus affecting the gastric artery severe stenosis of the hepatic artery. The mural thrombus also involved the entire origin of the superior mesenteric artery, with peripheral reperfusion. The renal arteries were similarly affected, although the inferior mesenteric artery was unaffected. The dissection extended to both iliac arteries, including the left internal iliac artery. The diagnosis was extensive Stanford type B (DeBakey III) aortic dissection involving major aortic branches (Figure 1).

Given the extent of the dissection and associated comorbidities, a two-stage hybrid approach was undertaken. The first stage involved supra-aortic vessel debranching via median sternotomy and inverted-T pericardiotomy. The brachiocephalic trunk, left common carotid artery, and left subclavian artery were ligated and subsequently reimplanted using a 14/7-mm Dacron bifurcated graft. This procedure was performed off-pump, with cerebral perfusion monitored by near-infrared spectroscopy (NIRS) (Figure 2).

The second stage consisted of thoracic endovascular aortic repair (TEVAR) with two overlapping Valiant stent grafts (36×100 mm and 36×32×150 mm), (Medtronic, Minneapolis, USA) to exclude the dissected segment and prevent false-lumen re-expansion (Figure 3).

Postoperatively, the patient had persistently elevated blood pressure initially managed with intravenous Nitroglycerin (PharmaZell, Germany), Nimodipine (Bayer AG, Germany), and Tachyben (Ever Pharma, Austria), later transitioned to oral therapy with Sevikar (Daiikhi-Sankyo, UK), Amlodipine (Sandoz GmbH, Switzerland), Tenaxum (Servier, France), Doxazosin (Pfizer GmbH, Germany), achieving partial control. Her postoperative course was favorable, with stable vital signs, no infection, and good wound healing. She was discharged in stable condition with dietary and monitoring recommendations and referred to the Cardiology Department for further re-

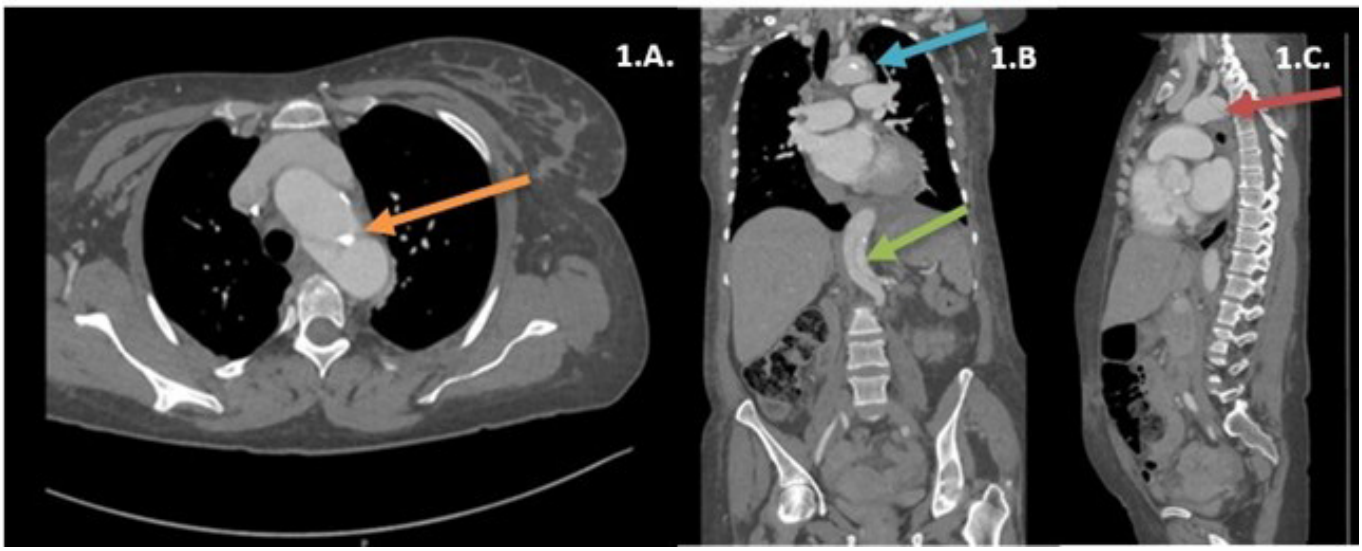

Fig. 1. Contrast-enhanced computed tomography (CT): **1.A.** (axial CT section) - The orange arrow highlights the aortic dissection flap at the level of the aortic arch. **1.B.** (coronal CT section) - The blue arrow indicates the propagation of the dissection along the thoracic aorta in the coronal plane; The green arrow points to the extension of the dissection into the abdominal aorta. **1.C.** (sagittal CT section) - The red arrow marks the dissection flap at the level of the aortic arch as visualized in the sagittal reconstruction.

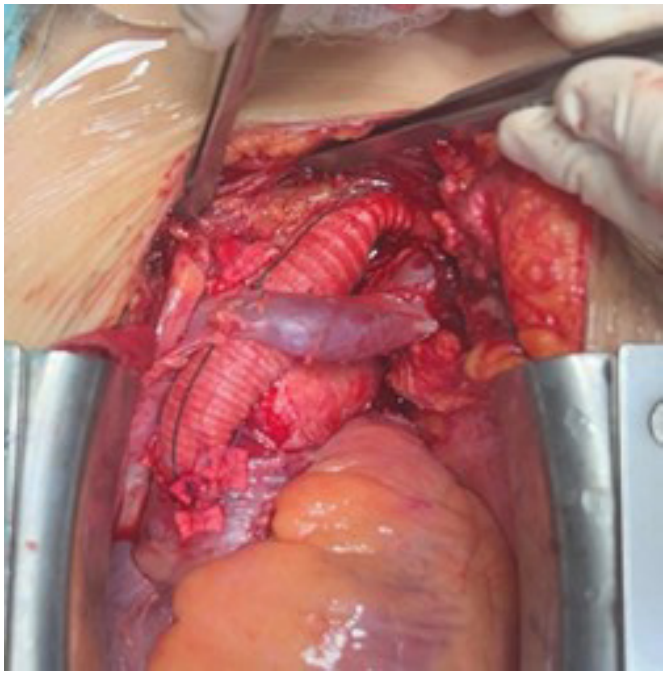

Fig. 2. Intraoperative view showing complete supra-aortic debranching performed with a single trifurcated graft, supplying the brachiocephalic trunk, left common carotid artery, and left subclavian artery from a common proximal anastomosis on the ascending aorta.

covery and antihypertensive adjustment. After two month the patient was readmitted to the cardiovascular surgery unit with severe chest pain radiating to the back, secondary hypertension of grade 3 refractory to medical therapy and multiple manifestations of systemic damage. Imaging studies confirmed the extension of the dissection proximally to the level of the aortic valve, with the ascending aorta measuring 49 mm in diameter. A partially thrombosed false lumen was observed in the descending thoracic aorta, along with a pericardial effusion of 7 mm, bilateral pleural effusion, cardiomegaly, and compressive atelectasis of the left lung (Figure 4).

We performed a redo sternotomy, extensive adhesiolysis, and replacement of the ascending aorta with a 32 mm Dacron graft and reimplantation of the supra-aortic vessels branches into the prosthetic graft (neo-aorta). Cardiopulmonary bypass time was 68 minutes and aortic cross-clamp time was 42 minutes. The procedure was performed under circulatory arrest, with antegrade selective cerebral perfusion employed to protect brain function; during this period, the right common carotid artery was clamped for 24 minutes and the brachiocephalic trunk for 20 minutes. (Figure 5).

Arterial cannulation was performed through the right femoral artery and brachiocephalic trunk. The patient's course was complicated by advanced chronic kidney disease that required permanent hemodialysis.

Postoperative echocardiography demonstrated concentric left ventricular hypertrophy with a 50% preserved ejection fraction, mild mitral and tricuspid regurgitation, and small collections of pericardial fluid. A follow-up CT angiography confirmed the correct positioning of the endoprostheses in the ascending aorta, aortic arch, and descending thoracic aorta, with evident supra-aortic branches and no signs of endoleak. The true lumen measured 27 mm at the arch and 18 mm in the descending thoracic aorta, while the false lumen measured 11-12 mm and contained thrombotic material. The dissection flap extended caudally beyond the stent graft to the level of the L4 vertebra, with both lumens opacified post-contrast beyond the origin of the celiac trunk.

During hospitalization, the patient presented a transient episode of temporo-spatial disorientation. The neurological consultation and cranial CT were immediately obtained, but no acute brain injuries were identified, and the episode resolved spontaneously.

Upon discharge the patient was afebrile, hemodynamically stable, with a heart rate of 80 bpm and a blood pres-

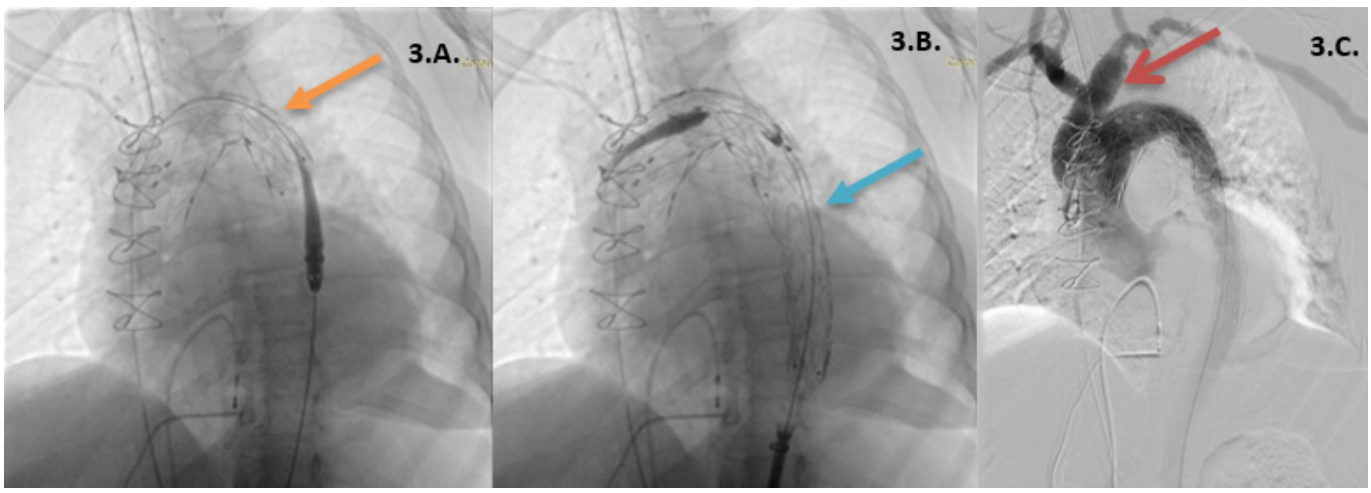

Fig. 3. Post-operative computed tomography angiography (CTA): **3.A.** (angiographic view) - The orange arrow indicates the TEVAR stent-graft deployed at the level of the aortic arch, demonstrating proper positioning and proximal sealing. **3.B.** (angiographic view) - The blue arrow highlights the distal portion of the stent-graft extending into the descending thoracic aorta, ensuring exclusion of the dissection in this segment. **3.C.** (angiographic view) - The red arrow marks the restored perfusion of the supra-aortic vessels achieved through the prior debranching procedure, with adequate filling of the reconstructed branches.

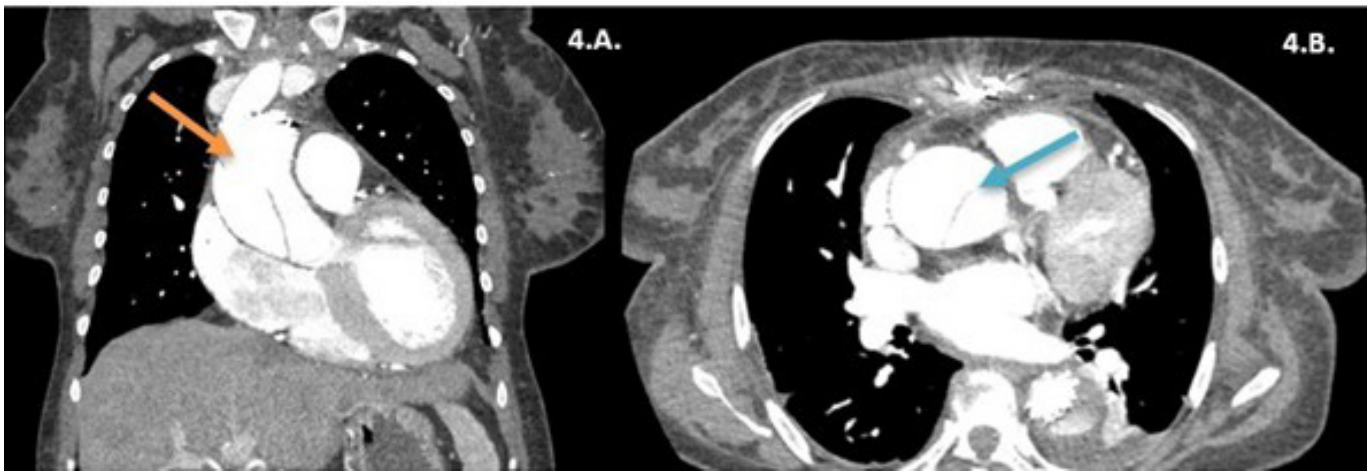

Fig. 4. Contrast-enhanced computed tomography (CT): **4.A.** (coronal CT section) - The orange arrow highlights the dissection flap within the ascending aorta as visualized in the coronal plane, showing clear separation of the true and false lumens. **4.B.** (axial CT section) - The blue arrow indicates the same aortic dissection flap in the axial plane, confirming its extension and the involvement of the ascending aorta.

sure of 143/71 mmHg and maintained sinus rhythm. The surgical wounds healed properly, with no signs of infection, and the neuropsychiatric condition was intact.

Discussion

Acute aortic dissection is a major clinical emergency with an estimated incidence of 3.5-14 per 100,000 person-years and hospital mortality of about 27%, which requires early diagnosis and rapid transfer of the patient to a specialized center equipped to manage such pathologies, where surgery is often crucial [1]. Acute aortic syndromes are a cardiovascular condition first described by Frank Nicholls in the autopsy report of King George II in the eighteenth century [8]. In the mid-twentieth century, the first clinically distinct variants of aortic dissection were identified, classified by DeBakey as type I and type II, originating from the ascending aorta, and type III, originating from the descending aorta [9]. In the 1970s, the Stanford classification appeared, further highlighting the different clinical practices in the management of ascending versus descending aortic dissection. According to the Stanford classification, dissections are grouped into type A, which involves the ascending aorta and requires immediate surgery, and type B (approximately one-third of all cases), located distal to the left subclavian artery, for which treatment can be both medical and endovascular [2,10].

The prevalent mechanisms proposed for acute aortic dissection include an intimal tear allowing blood to enter the media and create true and false lumina and an intramural hemorrhage of the vasa vasorum within the media that may subsequently communicate with the lumen. In both scenarios, severe hypertension and high pulsatile ejection forces facilitate hematoma propagation and longitudinal extension of the dissection [11–13].

The complexity of managing type B acute dissection continues to remain a challenge. The classic understanding has been that complicated type A and type B acute aortic dissections require surgical resection, while uncomplicated type B acute aortic dissection can be treated medically. Complicated type B dissection includes malperfusion indicated by impending organ failure, hypertension or persisting with elevated levels despite complete drug therapy.

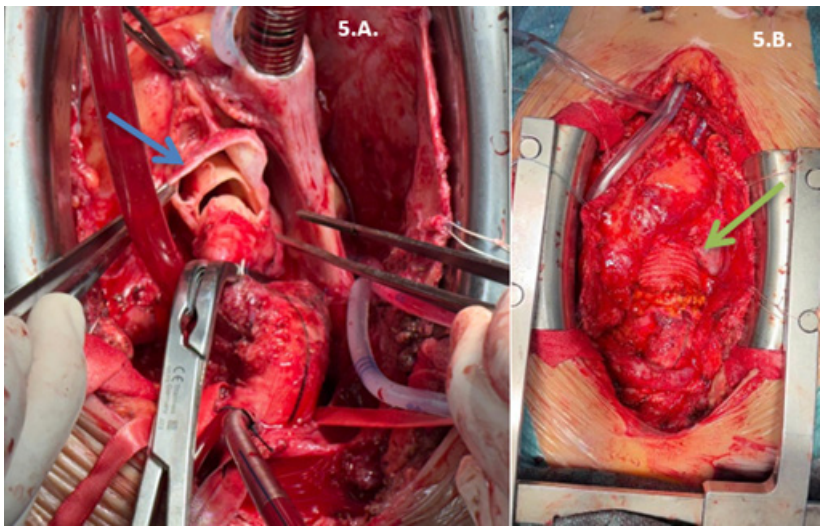

Fig. 5. Intraoperative view: **5.A.** The blue arrow points to the dissected segment of the ascending aorta identified during intraoperative evaluation. **5.B.** The green arrow denotes the ascending aortic prosthesis in situ at the end of the surgical intervention.

However, in cases where malperfusion may remain too subtle to cause obvious clinical symptoms, laboratory markers provide a sensitive method of detection. Increases in liver or pancreatic enzymes may similarly indicate mesenteric or celiac reduced blood flow, while an increase in creatinine would signify renal hypoperfusion. In addition, refractory pain can also be a clinical symptom that indicates organ ischemia [2]. Patients undergoing chronic dialysis are at extremely high cardiovascular risk due to factors such as treatment-resistant hypertension, widespread vascular calcification, and severe electrolyte imbalances [14]. These factors not only complicate the therapeutic approach and postoperative prognosis, but also significantly increase the risk of developing aortic dissection [15,16]. Atherosclerosis, especially when coexisting with severe hypertension, plays a significant role in the degeneration of the medial aortic layer, especially in elderly patients [7]. In addition, early-onset atherosclerosis leads to vessel fragility, and uremia-related coagulopathy, combined with the routine use of anticoagulants during dialysis, can lead to minor intramural hemorrhages that favor the progression of dissection through minimal intimal tears [17].

In patients on hemodialysis, both functional and structural assessment of the large arteries repeatedly demonstrate significant changes. Advanced imaging techniques such as high-resolution ultrasound with wall tracking, intima-medium thickness measurement, wall cross-sectional area, wall-to-lumen ratio, and pulse wave velocity provide an accurate assessment of arterial stiffness. These objective findings confirm that increased arterial stiffness and vascular remodeling play a central role in increased pressure load on the left ventricle and contribute to maladaptive cardiac remodeling [14].

In 2006, IRAD data reported a 3-year mortality for acute type B dissection close to 1 in 4. Most interestingly, it has been observed that patients with type A acute dissection usually have high early mortality rates, which stagnated after discharge. On the other hand, patients with acute type B dissection continue to have high rates of cumulative death, actually exceeding the rates of patients with acute type A dissection at 3 years, which is why our case was recommended for a rigorous postoperative follow-up after the hybrid intervention for type B dissection and prompt intervention in the case of the second readmission for Standford A type aortic dissection. Shock and renal failure have been identified as major predictors of mortality at 3 years. In 2010, using IRAD data, it was shown that patients with type B acute aortic dissection and refractory arterial hypertension (requiring ≥ 3 different classes of antihypertensive therapy at maximum tolerated doses, as in the case of our patient who was at the time of first discharge under quadruple antihypertensive therapy) showed a more than 20-fold increase in mortality in the case of medical treatment (35.6% *vs.* 1.5%; P=0.0003). Unfortunately, the alternative of open surgical treatment has a mortality rate in hospital of over 30% [18].

Despite continuous advances in perioperative strategies and surgical techniques tailored to patients' needs, the 30-day mortality rate in a cohort of 303 patients with thoracic or thoracoabdominal aneurysms is 21.3% (n=75) and higher for open surgery (n=41, 30.1%) than for TEVAR (n=34, 15.7%). Acute aortic dissection remains significant. End-stage renal disease patients undergoing chronic hemodialysis are known to have higher post-procedural morbidity and mortality rates compared to the general population, and their life expectancy is shorter. The average age of these patients was 65 years, and 64% were men. In the study conducted by Liang NL et al [16] suggests that people with chronic HD ESRD continue to have poor outcomes after intervention for any thoracic aortic pathology, both perioperative and long-term, with those with acute presentation tending towards higher mortality in open surgery (n=14, 31.8% vs. n=10, 15.7%; p=0.17). Historical and recent estimates of elective perioperative mortality range from 1.5% to 7%, as opposed to the estimate of this study which is 20% for patients on hemodialysis, the percentage difference being most likely represented by the inherent chronic condition of patients with end-stage chronic kidney disease, rather than the type of intervention performed. From 2005 to 2008, the United States Renal Data System identified 352 patients with ESRD on dialysis who underwent thoracic aortic repair, either by open surgery (n=136) or by thoracic endovascular aortic repair (TEVAR, n=216). Of these, 303 patients had thoracic or thoracoabdominal aneurysms, of which 47 (13.4%) had rupture. Aortic dissection was diagnosed in 44 patients (12.5%), and aortic trauma was reported in 5 patients (1.4%) [16].

The IRAD analysis led by Fattori et al. showed that although patients treated with TEVAR had complicated dissections more frequently (62% vs. 37%; P < 0.001), in-hospital and 1-year mortality was similar between groups, but 5-year mortality was significantly lower in patients with TEVAR compared to medical treatment (16% vs. 29%; P = 0.018). Similar results were confirmed in the INSTEAD-XL study [19], where TEVAR demonstrated a long-term survival benefit by reducing aortic mortality. In contrast, in the study by Liang NL et al. [16] the mortality rate at 30 days after thoracic/thoracoabdominal aneurysm repair was 21.3% (n=75/303), significantly higher for open surgery (30.1%) than for TEVAR (15.7%). In patients with end-stage chronic kidney disease on hemodialysis, perioperative mortality reached 20%, compared to historical estimates of 1.5–7%, suggesting that the poor prognosis is mainly related to comorbidities rather than the type of intervention [7].

Patients with end-stage chronic kidney disease on maintenance dialysis are a high-risk group for this condition. In dialysis-dependent patients, chronic kidney disease–mineral and bone disorder, frequently accompanied by secondary hyperparathyroidism, promotes medial vascular calcification and marked arterial stiffening. This increases pulsatile load and shear stress on the aortic wall,

facilitating acute aortic syndromes. Such patients often suffer from severe hypertension, which is difficult to control, despite regular dialysis and medical therapy [1,20]. Blood pressure control in aortic dissection represents a major therapeutic challenge, particularly in patients with treatment-resistant hypertension. Current guidelines recommend maintaining values below 135/80 mmHg in chronic cases, as adequate control remains central to the management of type B aortic dissection. Thoracic endovascular aortic repair (TEVAR) has been shown not only to stabilize the dissection but also to significantly reduce systolic and mean blood pressure, even in refractory hypertension. These hemodynamic improvements may lower the risk of subsequent complications and highlight TEVAR as an important adjunct to intensive care. Despite combination therapy, about 40% of patients continue to have severe hypertension, often requiring five or more classes of antihypertensive agents. Isolated systolic hypertension is frequent, reflecting increased aortic stiffness. Effective management therefore demands a comprehensive, multidisciplinary approach that considers the impact of comorbidities such as chronic kidney disease requiring hemodialysis and obesity, both of which further complicate blood pressure control [6,20–22].

While renal denervation has historically been used as a rescue strategy, baroreflex activation therapy (BAT) by stimulating the carotid sinus has recently emerged as an alternative. Case reports demonstrate that immediate activation of BAT, even in the context of a therapy-refractory hypertensive crisis after aortic dissection, can lead to a rapid and sustained reduction in blood pressure without affecting recovery. Although the evidence is limited, BAT can represent a promising rescue option in certain high-risk scenarios [23].

Even though emergency surgery is the agreed standard of care in type A dissections at Stanford, careful patient selection and timely intervention can significantly reduce mortality in type B dissections as well. Tools such as the GERAADA score have been developed to estimate postoperative mortality in patients with acute aortic dissection, providing a rapid and effective method of risk assessment based on basic clinical parameters. However, this score does not include certain high-impact risk factors, such as severe renal dysfunction or chronic dialysis, which the literature suggests may substantially increase perioperative risk [24].

The treatment of type B aortic dissection in the subacute phase is still debated. In the INSTEAD study, patients randomized to TEVAR between 2 and 52 weeks from baseline had a 5-year overall mortality of 11.1% compared to 19.3% in the medically treated group (P = 0.13), and aortic specific mortality was significantly lower, 6.9% versus 19.3% (P = 0.04). These results suggest that TEVAR, combined with medical therapy, may delay disease progression and improve long-term survival. Akin et al. proposed as indications for TEVAR in subacute or chronic type B dissection the presence of persistent chest pain, uncontrolled hypertension, an aortic diameter >55–60 mm or an annual growth rate >4 mm [2].

Dialysis patients with type A acute aortic dissection often experience arch entry ruptures and extensive vascular calcification, which requires more complex interventions, such as replacement of the arch with dacron prostheses and sometimes debranching procedures. This profile is associated with increased perioperative mortality (21% vs. 7%) and lower long-term survival (≈60% at 6 years). However, surgery remains justified, offering benefits even in a very high-risk cohort [7].

Dialysis patients undergoing proximal aortic surgery are one of the cohorts with the highest risk in cardiovascular practice. The large data in the registry show a perioperative mortality of 12.6% for aneurysms and 24.3% for type A dissections, with a 10-year mortality exceeding 80%, regardless of the indication. Old age, heart failure, and diabetes as a cause of ESRD were independent predictors of worse outcomes. Importantly, while elective repair of thoracic aneurysms in this population requires very careful patient selection, emergency surgery for acute type A dissection remains feasible and can save lives despite the increased risk [25].

In dialysis-dependent patients, thoracic aortic repair is associated with substantial and long-term perioperative risk. Comparative analyses show that TEVAR offers a clear short-term survival advantage over open repair, with lower mortality at 30 days and fewer respiratory complications. However, both approaches are characterized by poor long-term survival, with a one-year mortality rate approaching 50%. These findings highlight the extreme vulnerability of the ESRD population and highlight the need for careful selection of patients and timing of intervention [16].

Renal dysfunction significantly influences outcomes after thoracic endovascular aortic repair (TEVAR). Analyses of large databases demonstrate that while stages 1-3 of chronic kidney disease do not significantly increase early readmission or mortality, advanced CKD and especially end-stage renal disease (ESRD) are associated with higher mortality, longer hospitalizations, higher costs, and more frequent readmissions. These findings highlight ESRD as a key determinant of prognosis after TEVAR and underscore the need for tailored perioperative strategies in this high-risk population. tag [3].

Large-scale Medicare data show progressively poorer survival with advanced CKD, with one-year survival dropping from 78% in patients without kidney disease to just 48% in those with ESRD or on dialysis. Patients with advanced CKD or ESRD also face higher risks of sepsis and perioperative mortality, with 30-day reported mortality rates of up to 17% in the ESRD cohort. These findings underscore the need for careful patient selection, weighing the benefits of rupture prevention against the high morbidity and mortality expected in dialysis-dependent individuals [4].

Published outcomes of hybrid strategies are heterogeneous across techniques and patient profiles, and results from non-dialysis cohorts cannot be directly extrapolated to dialysis-dependent patients, who represent a distinct, higher-risk subgroup with different perioperative and long-term trajectories.

This hybrid one-step approach has demonstrated favorable morbidity and mortality profiles, although complications such as coagulopathy, neurological damage, and graft-related events remain concerning. The evolution of the device and refined techniques continue to improve their safety and applicability in complex arch diseases [26].

Modified triple-branched stent grafts have emerged as a promising surgical strategy for type A aortic dissection, providing a technically simplified alternative to conventional arch replacement. Ten-year institutional experience in nearly 500 patients has demonstrated low early mortality (4.8%), acceptable complication rates, and long-term sustainable survival exceeding 80% at six years. The high rates of false lumen thrombosis and the limited need for reoperation further support the safety and efficacy of this approach, although continuous surveillance of stent-related complications is necessary [27].

Great vessel debranching (GVD) was introduced as a means of extending the proximal landing zone to allow zone 0 TEVAR in patients with complex thoracic aortic disease. Clinical experience demonstrates high rates of patency of supra-aortic revascularizations and acceptable perioperative risks, with mortality below 10% and low rates of neurological complications. Medium-term survival remains moderate, but the durability of branch permeability supports GVD as a safe and effective adjunct to hybrid and endovascular arc repair strategies [28].

Diagnostic imaging in this population requires careful adaptation: contrast CT angiography remains central, but protocols should minimize exposure to nephrotoxic contrast, and alternative modalities, such as MRI angiography or contrast-enhanced ultrasound, may be preferable, especially for follow-up [29].

Conclusion

Acute aortic dissection in dialysis-dependent patients remains an uncommon yet highly lethal condition. The combination of end-stage chronic kidney disease, refractory hypertension, prior aortic surgery, and extensive vascular calcification creates a complex clinical scenario, increasing both perioperative risk and long-term mortality. Although outcomes are generally worse than in patients without renal failure, emergency or hybrid surgical–endovascular repair can be feasible and life-saving in selected cases.

Successful management relies on individualized treatment planning, meticulous perioperative care, and coordinated multidisciplinary follow-up. Continuous hemodynamic control and monitoring are crucial to prevent recurrence and improve survival in this high-risk population.

AI Tools Disclosure

Grammarly (v1.2.182.1722) was used for grammar and clarity improvements. All AI suggestions were reviewed and approved by the authors.

Authors' contribution

S-I. (Conceptualization; Writing – original sketch; Writing – revision and editing)
A. E.D. (Conceptualization; Supervision; Writing – revision and editing)
A. L. (Formal analysis; Investigation; Methodology)
H. M. M. (Conceptualization; Formal analysis; Methodology; Supervision; Writing – revision and editing).

Conflict of interest

None to declare.

Ethical Statement

Written informed consent was obtained from the patient's legal guardian for the publication of this case report and any accompanying images. This report complies with the ethical standards of the institutional research committee and the 1964 Declaration of Helsinki and its subsequent amendments.

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patient(s) to publish this paper.

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