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SCIENTIFIC ARTICLE - CASE REPORT

LATE DIAGNOSIS OF AORTIC DISSECTION (14 DAYS): ANESTHETIC CHALLENGES – CASE REPORT

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ABSTRACT

Introduction: Acute type A aortic dissection (ATAAD) is a highly lethal cardiovascular emergency, with mortality exceeding 50% within the first 24 hours if untreated. Early diagnosis is critical for prognosis, yet atypical clinical presentations frequently delay both suspicion and confirmation. **Case report:** An 80-year-old male with a history of systemic arterial hypertension, hypothyroidism, dyslipidemia, chronic hepatitis C, and epilepsy was initially admitted to a regional hospital with nonspecific symptoms of nausea, vomiting, and abdominal pain. During hospitalization, he developed seizures and aspiration, receiving empirical antibiotic therapy. Fourteen days after symptom onset, chest CT angiography revealed an intimal flap in the ascending aorta, consistent with type A dissection, and he was transferred to a referral center. He underwent surgical repair under target-controlled total intravenous anesthesia (TIVA-TCI), with multimodal monitoring and massive transfusion due to associated coagulopathy. Initially, he remained stable, with early extubation and progressive weaning from vasopressors. On postoperative day six, however, he developed septic shock caused by Pseudomonas aeruginosa, followed by progressive multiorgan failure and death. **Discussion:** The atypical presentation of ATAAD, with gastrointestinal and neurological manifestations, delayed diagnosis. Survival beyond seven days without surgical intervention is uncommon and was likely related to clot formation sealing the false lumen. Anesthetic management required slow, titrated induction, stable maintenance with TIVA-TCI, judicious use of vasopressors, and aggressive correction of coagulopathy, underscoring the complexity of such cases. Despite technically successful surgical repair, the outcome was unfavorable due to late infectious complications, consistent with the high morbidity and mortality reported in elderly patients undergoing prolonged cardiopulmonary bypass. **Conclusion:** This case highlights the importance of early clinical suspicion in atypical presentations, the pivotal role of the anesthesiologist in hemodynamic and hemostatic management, and the need for individualized strategies to optimize survival in cases of type A aortic dissection with delayed diagnosis.

Keywords: Aortic dissection, Anesthesia, Cardiac tamponade, Blood coagulation, Shock septic.

INTRODUCTION

Acute type A aortic dissection (ATAAD) is a rapidly progressive cardiovascular emergency associated with high lethality. It is characterized by a tear in the intimal layer of the aorta, usually in the ascending portion, allowing blood to enter the tunica media and form a false lumen.[1] Its incidence ranges from 5

to 30 cases per million inhabitants per year, with an estimated mortality of 1–2% per hour during the first 48 hours if untreated, reaching up to 50% within the first 24 hours.[2,3] The Stanford classification is the most widely used method for anatomical stratification, with type A defined as any dissection involving the ascending aorta, regardless of distal extension.[4]

Clinically, ATAAD typically presents with sudden, severe chest pain, often described as tearing or stabbing, which may radiate to the back, abdomen, or limbs, depending on the extent of the dissection. Many patients have a history of systemic arterial hypertension, a risk factor present in up to 80% of cases. Clinical presentation may be variable and nonspecific, ranging from acute neurological deficits, lower limb ischemia, and syncope to signs of cardiac tamponade or shock. Due to symptom overlap with other cardiovascular emergencies, the main differential diagnoses include acute coronary syndrome (ACS), pulmonary embolism, stroke, and ruptured aortic aneurysm. Diagnostic accuracy depends on a high index of clinical suspicion and the early use of imaging studies, particularly contrast-enhanced computed tomography, which is considered the gold standard for confirming the diagnosis and defining the extent of aortic involvement.[5] Early diagnosis is essential and may be supported by laboratory markers such as D-dimer, troponin, and NT-proBNP, in addition to imaging modalities such as contrast-enhanced computed tomography or transesophageal echocardiography.[6]

Cases of delayed diagnosis, such as the one reported herein, are rare and are usually associated with atypical clinical evolution or failures in the initial diagnostic workup. Survival beyond seven days without intervention is uncommon, and partial chronicity may lead to complications such as aortic rupture, aortic valve insufficiency, cardiac tamponade, and visceral or cerebral malperfusion syndromes.[7,8] Anesthetic management in ATAAD is complex, requiring strict blood pressure control, neurological protection, and specific strategies during cardiopulmonary bypass and circulatory arrest, including hypothermia and cerebral perfusion monitoring.[9] The presence of consumptive coagulopathy, inherent to the dissection process itself, is further exacerbated by cardiopulmonary bypass, necessitating careful replacement of coagulation factors and fibrinogen.[5] In addition, patients with ongoing dissection may develop progressive organ dysfunction (renal, respiratory, neurological), which makes anesthetic planning even more challenging, particularly in the setting of delayed diagnosis.[2,6] In this context, we report the case of a patient with ATAAD diagnosed 14 days after symptom onset, with atypical clinical evolution and complex anesthetic management, highlighting the strategies employed and the challenges encountered.

CASE REPORT

The patient was an 80-year-old male, 1.63 m tall and weighing 63 kg, with a medical history of systemic arterial hypertension, hypothyroidism, dyslipidemia, chronic hepatitis C, and epilepsy. His regular medications included olmesartan/hydrochlorothiazide 40/12.5 mg, amlodipine 5 mg, metoprolol 25 mg, levothyroxine 50 mcg, rosuvastatin 10 mg, and levetiracetam XR 500 mg. He had no known drug allergies.

The clinical course began 15 days prior to definitive hospitalization, when the patient sought medical care at a hospital in the southwestern region of Goiânia with nonspecific symptoms, including nausea, vomiting, and diffuse abdominal pain. Initial diagnostic hypotheses included urinary tract or gastrointestinal infection, and conservative clinical management was initiated. During hospitalization, the patient developed a generalized tonic–clonic seizure and was transferred to the intensive care unit (ICU), where he remained under neurological surveillance and supportive care.

Approximately seven days after admission, the patient experienced global clinical deterioration,

with a new seizure episode and an event of bronchoaspiration. In the context of an associated respiratory infectious condition, empirical antibiotic therapy with ceftriaxone and clindamycin was initiated. Due to persistent chest pain and progression to dyspnea, further investigation with chest computed tomography angiography was performed, which demonstrated a flap in the ascending aorta, highly suggestive of acute type A aortic dissection. The diagnosis was confirmed by an official radiology report, and the patient was immediately transferred to the Hospital do Coração de Goiás, a referral center for high-complexity cardiac surgery.

Upon admission to the ICU of the receiving institution, the patient was in poor general condition but conscious and oriented, breathing spontaneously with nasal cannula oxygen supplementation. Vital signs were abnormal, with a blood pressure of 182 × 110 mmHg and a heart rate of 110 beats per minute. Initial stabilization was undertaken, and surgery was scheduled for the following morning under a controlled urgent setting.

In the operating room, the patient was transferred on a stretcher with active thermal blanket support and underwent standard monitoring, including continuous electrocardiography, pulse oximetry, and noninvasive blood pressure measurement. A large-bore peripheral venous access (16G) was obtained in the left upper limb under ultrasound guidance. Right radial arterial cannulation was performed with an 18G catheter for continuous invasive blood pressure monitoring. Airway assessment was not performed prior to anesthetic induction. Definitive central venous access was established by the surgical team. Additional monitoring included anesthetic depth sensors, capnography, and a nasal temperature probe, all placed after tracheal intubation.

Antibiotic prophylaxis was administered with cefuroxime 1.5 g, with an intraoperative reinforcement dose of 750 mg. Anesthetic induction was performed gradually with strict hemodynamic control, using sufentanil 30 mcg, midazolam 15 mg, and rocuronium 50 mg. Orotracheal intubation was achieved using videolaryngoscopy (Cormack–Lehane grade 2A), with placement of an 8.0-mm endotracheal tube on the first attempt, without complications. Anesthetic maintenance was provided with propofol using a target-controlled infusion system (TIVA-TCI), oxygen at 2 L/min, and intermittent boluses of rocuronium, midazolam, and sufentanil as clinically indicated.

As intraoperative adjuvants, hydrocortisone 500 mg, dexamethasone 10 mg, calcium gluconate (one ampoule), epsilon-aminocaproic acid 4 g, vitamin C (four ampoules), and metaraminol as needed for blood pressure support were administered.

During the surgical procedure, cardiac tamponade was identified (Figure 1), along with a large clot at the site of the dissection (Figures 2 and 3), which, according to the surgical team, was a determining factor in containing bleeding and preserving the patient's life until transfer and definitive repair of the aortic dissection (Figure 4). Reconstruction of the ascending aorta was performed using cardiopulmonary bypass (CPB), with CPB and aortic cross-clamp times of 240 and 149 minutes, respectively. The total surgical time was 8.5 hours.

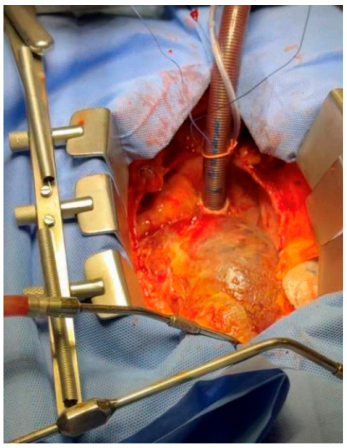

Figure 1. Tamponed pericardium.

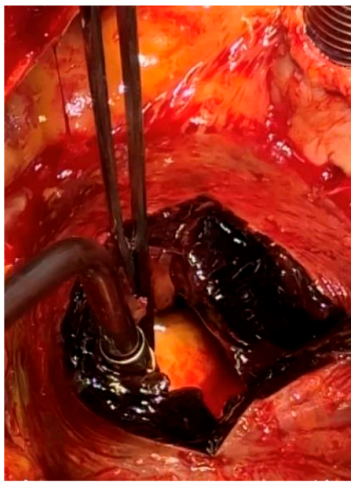

Figure 2. Formation of clots near aortic dissection.

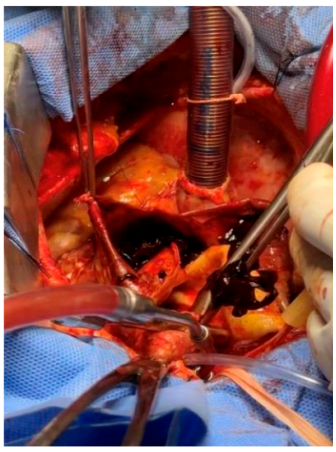

Figure 3. Removal of clots near an aortic dissection.

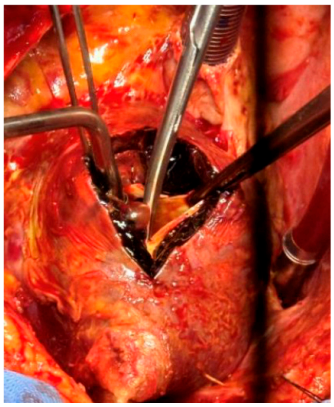

Figure 4. Access to the dissected aorta.

Total intraoperative urine output was 400 mL, with a positive fluid balance of 1,662 mL. The estimated blood balance was negative by 800 mL, and volume replacement was performed with 4 L of Plasma-Lyte®, seven units of leukoreduced filtered packed red blood cells, four units of fresh frozen plasma, nine units of cryoprecipitate, and one pool of leukoreduced random donor platelets (five units).

At the end of the procedure, the patient was transferred intubated to the intensive care unit (ICU), under assisted mechanical ventilation and continuous infusion of vasoactive agents: dobutamine (2.64 mcg/kg/min) and norepinephrine (0.17 mcg/kg/min), with hemodynamic stability and light sedation (RASS −1).

The patient remained intubated for 8.5 hours in the immediate postoperative period and was successfully extubated in a gradual manner, with maintenance of vasoactive amines and subsequent weaning according to clinical response. He progressed satisfactorily until postoperative day six, when he developed hemodynamic instability, decreased level of consciousness, and signs suggestive of septic shock. He was reintubated, and intensive supportive care was reinstated. Subsequent cultures isolated Pseudomonas aeruginosa, and targeted antimicrobial therapy was initiated. Despite the measures adopted, the patient progressed to progressive organ failure and died nine days after surgery.

DISCUSSION

ATAAD represents a cardiovascular emergency with high lethality, in which survival is intrinsically linked to the rapidity of diagnosis and surgical intervention. The delayed clinical presentation observed in the present case, occurring 14 days after symptom onset, is a rare and atypical finding. The literature reports an early mortality rate for untreated patients that may reach 50% within the first 24 hours and exceed 80% within two weeks, underscoring the exceptional nature of this patient's survival over such a prolonged period.[9]

The patient's initial presentation, which included gastrointestinal and neurological symptoms (seizure episodes), diverted diagnostic attention away from cardiovascular causes. This observation is consistent with extensive literature describing the atypical clinical presentation of ATAAD, particularly in elderly patients and those with comorbidities, which may include mesenteric ischemia, focal neurological deficits, or absence of classic chest pain.[1,9] In such scenarios, computed

tomography angiography of the chest and abdomen emerges as an indispensable diagnostic tool. The incorporation of biomarkers such as D-dimer and troponins, although useful for initial screening, does not replace imaging confirmation.[2]

The patient's hemodynamic stability, maintained for a prolonged period prior to surgical intervention, suggests the formation of a clot within the false lumen, acting as a natural "plug" that temporarily limited progression of the dissection. However, such stability is inherently precarious and does not eliminate the need for immediate surgical correction, given the imminent risk of rupture.[7]

From an anesthetic standpoint, the management of patients with ATAAD poses significant challenges. Perioperative care requires extreme caution to avoid abrupt changes in blood pressure that could exacerbate aortic injury. A slow, titrated anesthetic induction using hypnotic agents, potent opioids, and neuromuscular blockers proved effective in preserving hemodynamic stability in the present case. Anesthetic maintenance with TIVA-TCI combined with vasopressor support was appropriate for hemodynamic control and protection of target organs.[4]

Invasive and multimodal monitoring, including cerebral oximetry, capnography, and anesthetic depth monitoring, was crucial to guide hemodynamic management and ensure adequate tissue perfusion, particularly during the pre–cardiopulmonary bypass (CPB) phase. Judicious use of vasopressors, such as metaraminol, was essential to maintain arterial pressure at levels sufficient to ensure perfusion of vital organs, including the brain and kidneys.[8]

Consumptive coagulopathy, a common pathophysiological phenomenon in ATAAD, is exacerbated by CPB, requiring rigorous hemostatic management. Exposure of the aortic subendothelium and platelet activation induce coagulation disturbances that necessitate volume resuscitation and replacement of blood components guided by laboratory and clinical parameters.[5] In the present case, the need for massive transfusion of packed red blood cells, plasma, cryoprecipitate, and platelets, in addition to the administration of aminocaproic acid, underscores the severity of the associated hemostatic disorder.

Despite an initially favorable recovery and early extubation, the patient developed septic shock due to Pseudomonas aeruginosa on postoperative day six, resulting in multiple organ failure and death. This outcome is consistent with the literature, which identifies late septic shock as one of the leading causes of in-hospital mortality following surgical repair of ATAAD.[2] Factors such as advanced age and prolonged cardiopulmonary bypass time may have contributed to postoperative morbidity and mortality.

CONCLUSION

The present case highlights the importance of maintaining a high index of clinical suspicion for the diagnosis of acute type A aortic dissection in atypical and delayed presentations. This report reinforces the strategic role of the anesthesiologist throughout all phases of perioperative care, emphasizing the need for individualized planning, rigorous monitoring, and a dynamic approach to mitigate risks and optimize patient survival in such a challenging condition.

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